Virginia MEDICAL MONTHLY

OFFICIAL PUBLICATION OF THE MEDICAL SOCIETY OF VIRGINIA

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Medical Society of Virginia
Annual Session – Richmond
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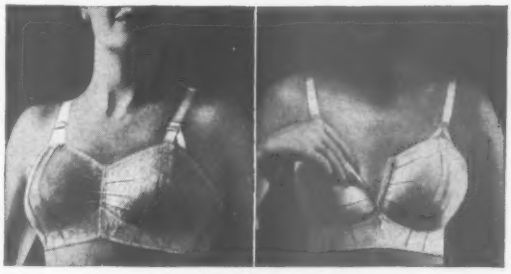

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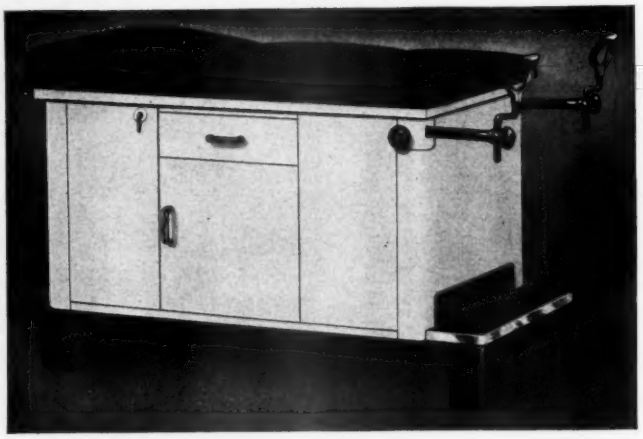

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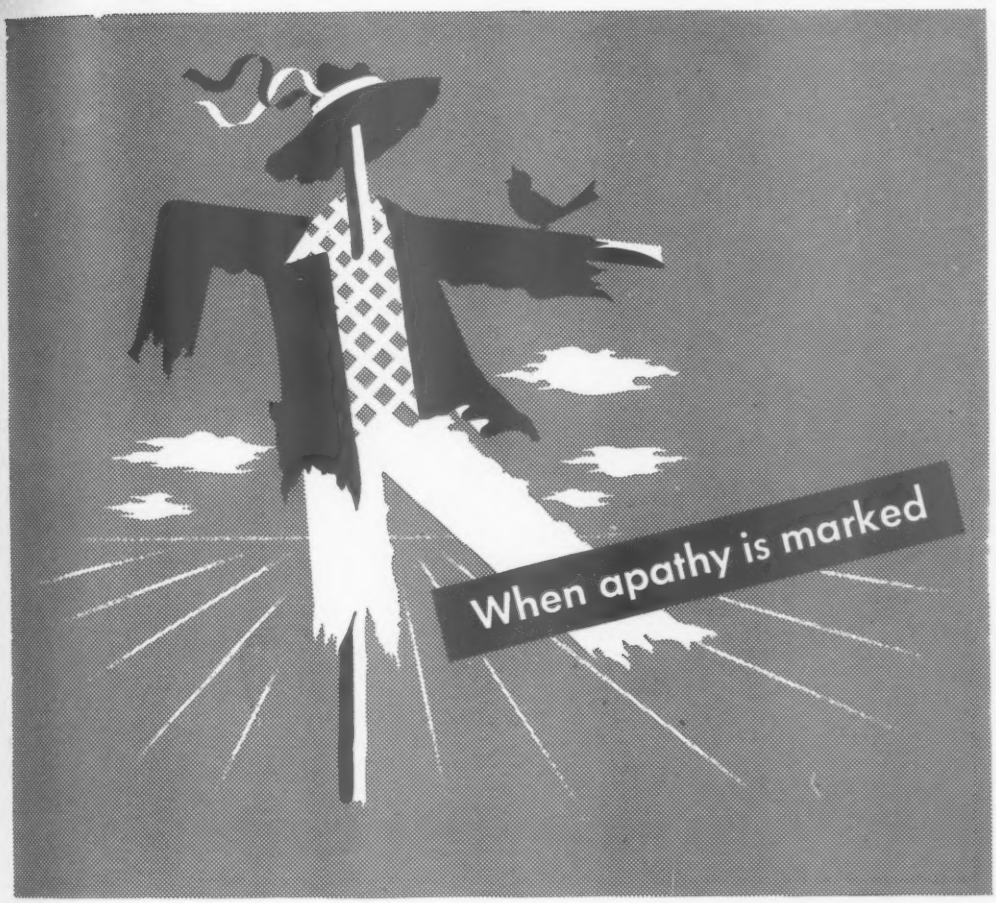

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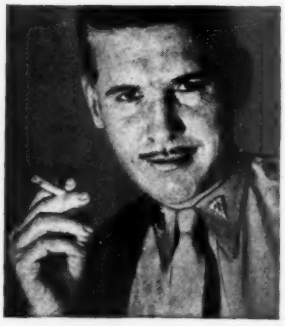

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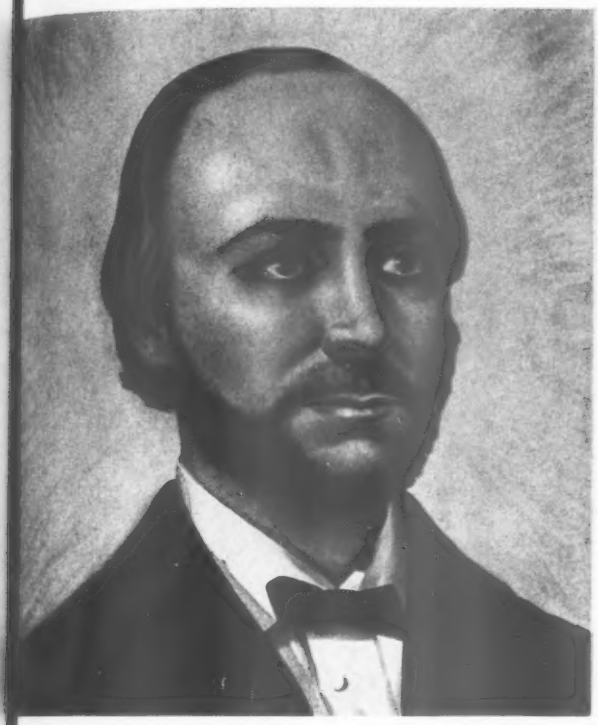

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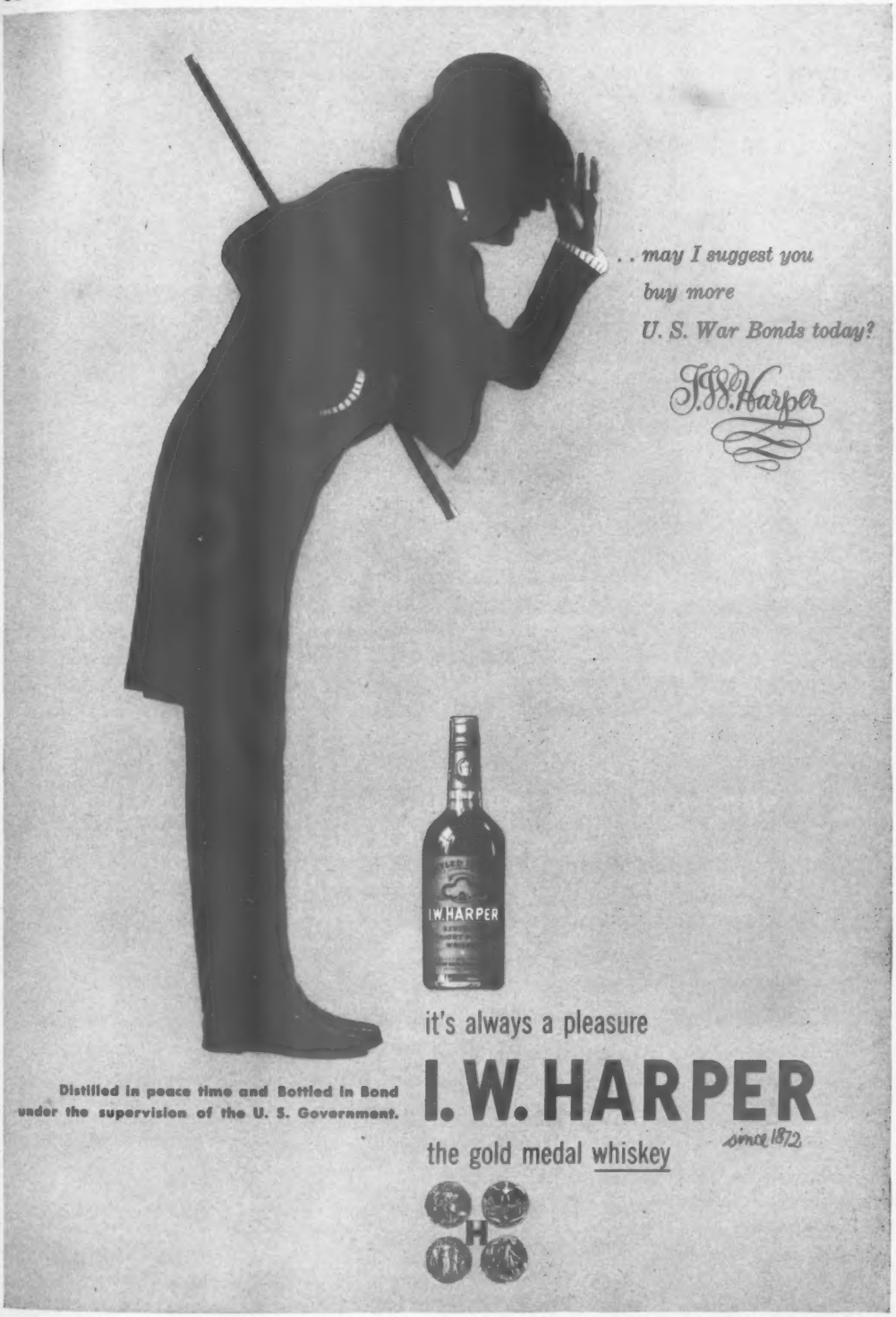
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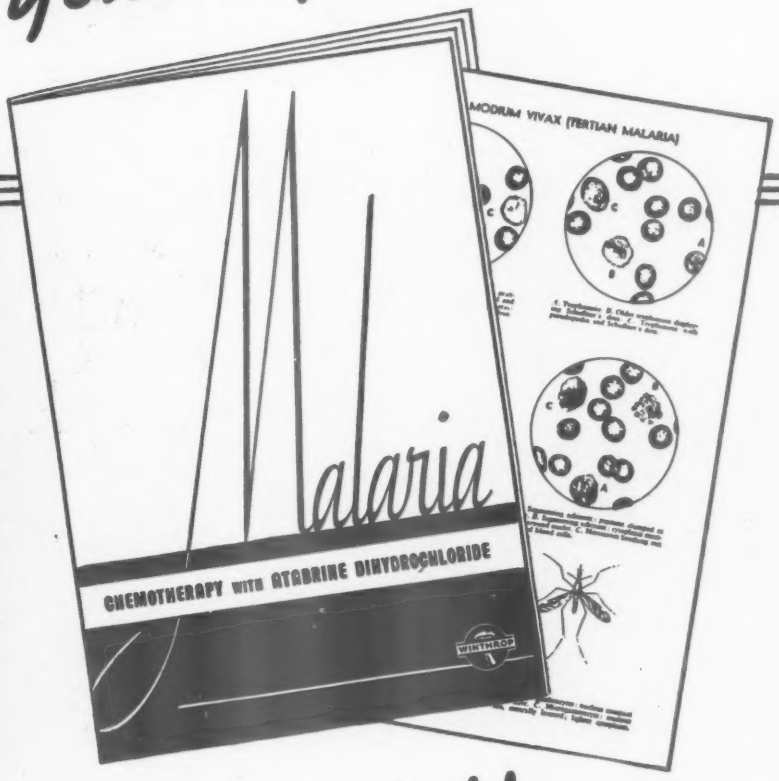

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Guest Editorial

Little Nazi-Made Zombies

I HAPPENED to be in Europe during the summer of 1926. In France and Belgium it was evident everywhere one went that there was a dearth of middle-aged men. Women were managing factories and stores and occupying other business positions usually held by men. This made me wonder about the status of the children born during the first World War, who were then from eight to twelve years of age. But few of them were seen upon the streets, and those who were casually observed appeared to be well and happy. The others, orphans or otherwise, were probably in schools, and no definite investigation of them was made by me at that time.

During the summer of 1931, I attended the International Neurological Congress in Berne, Switzerland, and I determined to inquire as well as I could into the condition of the first World War children who were then adolescents from thirteen to seventeen years of age. French, Dutch, Swiss, Belgian, and English neuropsychiatrists, more or less familiar with the situation, were interviewed. I was astonished to find that in their opinion these children, born during a period of horror, distress, massacre, and rape, were about as normal mentally and physically as those born during the piping times of peace.

Later more superficial inquiry was made of those who had visited Spain since the Civil War, concerning the young children born during that period. These children seemed to be below par physically and to have less high spirits than normal children. This condition was attributed to mal-nutrition because of the rapid improvement made when they were better fed.

And now we have a most interesting report of the children of Nazi-occupied Europe from 1939 through the school period of 1941. In a paper in *Psychosomatic Medicine*, July 1943, Marie Helen Mercier, M.D., and J. Louise Despert, M.D., state among other observations that shortly after the beginning of the scarcity of food, an increase in delinquency and stealing was noted, especially in adolescents. The children who had begun to steal through necessity, soon began to take things of no use whatsoever. Statistics computed on 500 children indicated that more than half showed signs of nutritional deficiency. Children who were observed in foster homes, these investigators said, showed indications of deep-seated (psychic) trauma though many presented no peculiarity except that "they lacked gaiety and enthusiasm and *they never smiled.*" Think of it, no bright eyes shining, no merry laughter, none of youth's spontaneity.

The question of the effect of a global war upon its generation of children is, of course, important, but the question of its effect upon children of the Nazi- occupied countries is of paramount importance. These, with the children of unoccupied countries, shall have the burden and responsibility of reorganizing the world that is to be, and at the same time, they shall have the greatest opportunity ever given a generation.

What are we doing and what shall we do to see that they shall not be found wanting? The answer is not easy or specific. No one mind, the mind of no one nation and of no one race, can truly comprehend, much less solve the problem. It is not a matter merely of vitamin feeding plus some inelastic rules plus preconceived charts and graphs, or plus anything else. The planning must be international and interracial, and to be everlasting, must be everchanging.

Let the statesman and the mechanic, the business executive and the laborer, the economist and the educator, the lawyer and the prelate do what can be done with their special abilities, and still there looms large the need for those who through love of children, and those who because of a knowledge of eugenics, obstetrics, paediatrics, psychology, psychiatry, child-guidance, and social service, to help the oncoming and ever oncoming generation.

We shall need an interglobal wise, unselfish organization of those who can speak a common language and have a common purpose which will receive the common support of the peoples of the earth. Such an organization is no more impossible than was the birth and fruition of the Red Cross.

I close by asking, shall we doom the future by our present inertia? It seems a puerile expression of our adulthood to foster a world of little zombies.

BEVERLEY R. TUCKER, M.D.
Richmond, Virginia.

Floral Eponym (15)

PAEON

Paeon (Paean or Paiëon) was classed by Homer among the Olympian gods of whom he was, as his name implies, the "healer". Later the name was applied to Aesculapius, then to any god who might repair or avert evil of any kind, as for instance, to Apollo. Paeans were chants in honor of Apollo, sung to deprecate misfortune in battle or to avert disease.

When Mars was wounded by Diomede, he bellowed with pain and betook himself to Olympus. Jove sternly reprimanded him and bade Paeon, the family physician, heal him, which he did.

The genus, Peony, the gorgeous flower which brightens our gardens in the Spring, is named for him.

CORRECTION OF MULTIPLE DEFORMITIES OF NOSE*

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Because the nose protrudes from the center of the face any deformity connected with it may be conspicuous. Malformations of appreciable degree involving the external nose can interfere with the social and economic life of the individual while deformities of the internal nose may seriously impair nasal function. The external appearance of the nose is important, but it is of less importance than is the fundamental consideration of its function. Adequate breathing space is very necessary because it promotes good drainage and proper ventilation of the respiratory tract which are conducive to normal development and good health. An external deformity is often combined with partial or total obstruction internally. The septum may bulge into both nasal passages, interfere with normal breathing and cause the external nose to deviate from the midline of the face.

In the long convex nose with a drooping tip and subluxation of the lower part of the septum obstructed breathing may be pronounced. Here the chief factor in bringing about the obstruction does not lie in the malformation of the septum alone but in the overhanging tip. Until the tip is elevated, the greatest source of obstruction will still remain. Only when the septal operation is combined with that of lifting the tip can the obstruction be relieved. There are other cases in which there is a marked drooping of the extreme tip of the nose with a short columella without deviation of the nasal septum. These cases do not require a septal operation. Lengthening the columella and lifting the tip will suffice to relieve the obstructed breathing.

Knowledge of the normal bony and cartilaginous framework of the nose is necessary to a full understanding of the multiple deformities of the inner and the outer nose. The soft structures are supported by a skeleton of bones and cartilage symmetrically arranged. The upper third of the external support is composed of two nasal bones which meet to form the bridge, the perpendicular plate of the ethmoidal bone, and the nasal processes of the maxillae. The remainder of the external support consists of the upper and lower lateral cartilages, the triangular cartilage of the septum and the columella. The internal nose is divided into two compartments by the septum which is composed of bone and cartilage. An injury which dislocates either the external or internal bone or cartilage may result in external deformity, interference with breathing and prevent proper drainage of the nasal secretions.

Many nasal deformities are caused by fractures which occur in early life. The very small child while learning to walk and the older child at play may sustain a firm blow on the nose. This may result in a fracture which will often interfere with the normal development of the nose if displaced parts do not receive proper attention at the time of injury or shortly following it. As a result of such an injury partial or complete nasal obstruction may follow and because of the obstruction the child's general condition will often suffer. There may be underdevelopment of the chest with a tendency to upper and lower respiratory infection. The necessity of early repair is obviously very important. A fracture of the nose, as of any other part of the body, should be reduced as soon as possible. This should be done preferably within forty-eight hours of the accident to prevent deformity and preserve normal function, provided there is no coexisting injury such as frac-

Profile view before operation. Elongated nose with depression immediately above tip. The breathing passages are obstructed due to thickened and deflected septum.

Profile view after operation. Resection of the thickened and deviated septum with slight lifting of the tip produced a good functioning nose. A cartilaginous graft which filled in the depressed area corrected the external deformity.

*From the Department of Surgery (Otorhinolaryngology), New York Polyclinic Medical School and Hospital.

Profile view before operation. Convex dorsum with drooping tip. A thickened and scoliosed septum, together with the drooping tip produces impaired nasal breathing.

Profile view after operation. Correction of external deformity improved the appearance of the nose. Resection of the cartilaginous and bony septum with lifting of the tip produced a normally functioning nose.

Full view before operation. The drooping tip shortens the upper lip, and interferes with normal breathing.

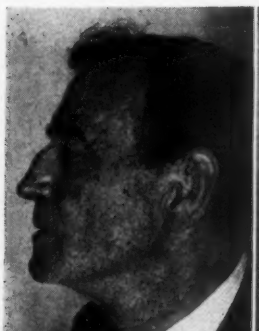
Full view after operation. The lifted tip facilitates proper breathing and lengthens the upper lip.

Profile view before operation. Humped nose with deviated and hanging septum. Nasal breathing obstructed.

Profile view after operation. Resection of the nasal septum and lifting the tip produced a good functional result, while lowering the bridge, narrowing and shortening the nose improved its appearance.

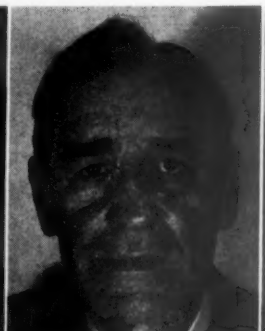
Full view before operation. The elongated tip interferes with normal breathing, shortens the upper lip and spoils the appearance of the mouth.

Full view after operation. The combined internal and external correction improved the function of the nose as well as its appearance.



Profile view before operation. Depression of lower third of nose which resulted from an infected hematoma of septum due to trauma.

Profile after operation. Cartilage and bone from the deviated septum filled in depression after the nose was straightened, narrowed and lifted.



Full view before operation. Note the scoliotic and depressed appearance of the dorsum.

Full view after operation. The deviated and depressed dorsum is straight and function of nose is restored.

ture of the skull, severe shock, or neurological damage. Any of these should preclude an attempt at early repair.

With reference to the correction of the long-standing deformity, whatever the defect, it must be analyzed with proper regard to the accepted criteria of

Profile view before operation. Elongated convex nose with sharp pointed tip. Breathing is markedly obstructed due to drooping tip. Note how half of the nostril is obstructed because of this drooping. The nasal septum is straight.

After operation. Removing the convex dorsum with shortening and lifting the tip produced a good functioning nose and improved its appearance.

form and proportion. The removal of excess tissue or the supply of structural deficiency must conform to the requirements which will result in the type of nose suitable to the outline of the face. Not infrequently the angle of the nose to the face is a determining factor in the facial harmony.

The operative procedure required depends upon the deformity to be corrected. However, if the skin is intact the operation is usually planned so that external incisions will not be required.

The anatomical relationship between the inner and outer nose is so intimate that deformities and malformations of the external nose frequently affect its function as well as its contour. Though this relationship is understood, it seems necessary to stress the need of correcting the inner and outer defects of the nose in one and the same operation. For instance, when an injury to the nose is sustained which results in a deflected septum and a sagging of the bridge (saddle nose) a submucous resection of the septum is required. The cartilage and bone, which has been removed from the septum, should be utilized to serve as the graft to fill in the depression on the dorsum of the nose. In patients with this type of combined inner and outer injury to the nose with external deformity and obstructed nasal breathing of long standing, not only should the intranasal obstruction be relieved but attention should be given simultaneously to the correction of the external deformity for the relief of the obstruction. If the external correction is not included in the operative procedure, a second operation is required to build up the depression. This operation will necessarily have to be more extensive because the cartilage and bone from the septum will no longer be available for the graft. Another part of the body will then have to be utilized to supply the graft which is usually taken from an ear, a rib, the crest of the ilium or from a donor.

Attention is directed to a number of conditions in which the surgery of the inner and the outer nose can and should be combined. Aesthetic and functional results can be had in the same operation in such conditions as: (a) old fractures with displacement; (b) depressed or saddle nose; (c) developmental defects. The depressed or saddle nose which follows nasal injury frequently results from the breaking down of an infected septal hematoma. If the hematoma is emptied shortly after its formation,

Before operation. Convex nose with obstructed breathing due to deviated septum.

After operation. Septum resection and correction of external deformity resulted in a good functioning nose and an improvement in its appearance.

infection is less likely to occur. Septal abscesses, resulting from the formation of a hematoma, may in very rare instances produce meningitis and this possibility should be kept in mind.

Most nasal malformations are congenital, developmental or traumatic. A small number is due to disease. The external deformities most frequently seen are the humped or hooked, the twisted or deflected,

Profile view before operation. Elongated convex nose with tucked in tip. Nasal breathing is obstructed due to deviation of the septum and depressed tip.

Profile view after operation. Lowering of the bridge and shortening the nose improved the patient's appearance while correcting the septal obstruction and lifting the tip produced good function.

Full view before operation. The drooping tip and deviated septum interfere with breathing.

Full view after operation. The deviated septum has been resected, the convex dorsum removed, and the tip elevated. Good function and improved appearance is the result.

the depressed or saddle nose, and the large and bulbous tip with malformations of the columella. In correcting these conditions consideration should be given to the plan of procedure to include a good functional, as well as a good cosmetic, result. The procedure will necessarily have to vary, depending upon the type of deformity. Adequate preparation includes a checkup on the general condition of the patient, an examination of the sinuses and a thorough planning of the operation. In scheduling the operation, sufficient time should be allowed as these operations are tedious and time consuming. To obtain the desired result, forethought, ingenuity, patience, and strict attention to details are most essential.

The obstructed nose, due to a thick deviated septum with marked sinking of the bridge, is a type that will require correction of the septal deformity and filling-in of the dorsal defect, utilizing the removed cartilage or bone as a graft. The humped or hooked nose, with a twist and deflection of the tip, is corrected by sawing or chiseling away the hump, sawing through the nasal processes of the superior maxillae, shortening the septum, adjusting the bony and cartilaginous structures and lifting the tip. In the correction of the large and bulbous tip with malformations of the columella, the amount of triangular and lower lateral cartilages requiring adjustment and removal will depend upon the shape and outline of the nasal orifices. There are a variety of these deformities and in each operative procedure it will be necessary to conform to the requirements of the individual case. Sound surgical judgment is essential and there are few fields of surgery in which an artistic sense is so much needed. This is especially exemplified in those cases of deformity which sometimes result in scar tissue formation due to folliculitis of the nasal orifices.

Profile view before operation. High dorsal convexity.

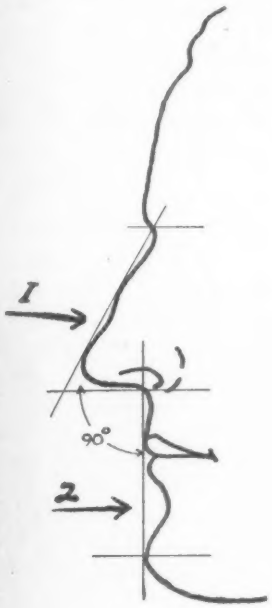
Profile view after operation. Dorsum lowered.

Profile view before operation. Very high convex dorsum with sharp pointed tip and hanging septum.

Profile view after operation. Dorsum lowered, septum shortened and tip lifted and rounded.

The photographs shown will assist in helping to visualize the type of case that requires a combined operation on the inner and the outer nose.

In the diagram below the septolabial angle indicated by the curved line drawn from the center of the tip of the nose to the center of the closed lips varies



PROFILE VIEW OF THE FACE

from 90 to 105 degrees. The long nose will shorten this angle and cause the tip of the nose to overhang the upper lip, spoil the appearance of the mouth and interfere with breathing. A very short nose increases the angle with a marked accentuation of the upper lip. The best measurement is about 90 degrees.

Another mathematical guide which may be used to determine the proper length of the nose can be arrived at through two simple measurements:

(1) A line from the root of the nose to its tip.

(2) A line from the base of the columella to the center or apex of the chin.

Line (1) and line (2) should be equal lengths or line (1) slightly shorter, depending upon the vertical dimension of the chin.

COMMENT

In addition to the satisfactory functional results obtained in the correction of nasal malformations (internal and external) new avenues of personal and professional experiences are opened to the patient. The entire outlook on life is often improved, and particularly is this true in the early and middle aged groups. An otherwise good featured face may be spoiled by a conspicuous deformity of the nose. These patients are often hypersensitive and introverted. The good results, which can be had in this field of corrective surgery, are well worth the time and effort. They are: improvement in the function of the nose which should make less likely the development of respiratory infections, improvement in the appearance of the patient, and the elevation of morale with enhancement of personality to the degree that the individual finds it less difficult to make proper social and business contacts.

CONCLUDING COMMENT

The necessity of combining surgery of the outer nose with that of the inner nose is essential to the proper correction of certain multiple deformities of the nose. This combined procedure, when properly executed, will result in a better functioning respiratory tract and an improvement in the appearance and mental state of the patient.

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ACUTE SURGICAL CONDITIONS COMPLICATING MALIGNANCY*

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From time immemorial the word "cancer" has carried a sinister significance. To the general public it is an absolutely hopeless prognosis. It means a long drawn out wasting disease, with weeks, and sometimes months, of suffering: a disease for which there is no ultimate relief other than death. Many of our professional brethren have the same feeling of utter hopelessness, and that, in spite of the fact that several years ago the American College of Surgeons sponsored a "Cured Cancer Club". By cured they meant a patient who was entirely free from any evidence of the disease five years after it had first been diagnosed and treated. To become a member of this club one must have pathological evidence that the original growth was cancer. No clinical diagnosis was accepted regardless of how self evident the condition was. The club now numbers several thousand individuals who have met all of these requirements.

The change in the treatment of cancer in the last few years has added something to this hopeful outlook. The addition of radium and x-ray has saved many lives that would have been considered hopeless a few yers ago.

We now know that cancers of the breast carry from 25 to 75 per cent five year cures, depending upon whether there is axillary involvement or whether the tumor is limited to the breast. Cancer of the cervix has almost as high a salvage if treated with radium and x-ray alone and surgery is not attempted. We have long known that skin cancers, in nearly 85 per cent of the cases, will respond to radium, x-ray, surgery or electrical coagulation if seen while they are still limited to the original lesion and metastases have not occurred.

With these statistics well known, there is no excuse for anyone to take the position that if an acute surgical condition superimposes itself upon a cancer victim, even though the cancer is still active, there is no reason for treating the condition because the patient is hopelessly ill anyway. With this in mind, I have been prompted to report a few clinical histories to prove that it is worth while to overlook the fact that the patient has, or has had, cancer, and treat the acute condition as though the patient were an otherwise normal individual.

Mrs. C. W., age 28 years. First seen in the office in 1935 when she was 22 years old with an erosion on the cervix which was cauterized. She had one child and had had an abdominal pelvic operation two years before. She was sent back in the office in 1941 with an erosion which involved almost the entire cervix. The uterus was not enlarged and there was no fixation. The pathologist reported the biopsy as a squamous cell carcinoma; clinically it was a Schmidt 2. She was given 5,716 mgm. hours of radium in March, 1941. Following this she began to run a high temperature and was given transfusions and sulfathiazole without any influence on her temperature. She had a mass in the right side of her pelvis with no tendency to localize in the cul-de-sac. It was felt that the mass was definitely in the folds of the broad ligament and she was sent home for observation. On May 9, 1941, she was returned to the hospital and an exploratory operation was performed. A large tubo-ovarian abscess was found and the right tube and ovary were removed. Apparently the left tube and ovary had been removed at the first operation. From June 19 to August 18, 1941, she was given 10,500 "r" over four 8 x 10 pelvic portals. She has been kept under fairly constant observation ever since and has shown no evidence of recurrence of her carcinoma—that, in spite of the fact that she was only 28 years old when the diagnosis was first made.

Mrs. R. B. B., white, age 52 years. First seen October 27, 1942, with a squamous cell carcinoma of the cervix, Schmidt 1. She was given 7,560 mgm. hours of radium. As it was such an early case she was not immediately given deep x-ray therapy, but from January 5 to March 17, 1943, she was given 10,000 "r" through four 8 x 10 pelvic portals. On March 26 she complained of an acutely tender mass in the right side of her pelvis. Her W. B. C. was 10,050; PMN 73 per cent. On April 6, 1943, the mass was explored and proved to be a ruptured appendiceal abscess. The pus was evacuated; the abscess cavity washed out, dusted with sulfanilamide powder and the abdomen closed without drainage. She was last seen on August 17,

*Read before the annual meeting of the Medical Society of Virginia, at Roanoke, October 25-27, 1943.

1943, when she was apparently entirely well. It has been less than a year since she was first seen, so we can make no claim for cure of the cancer. What we are claiming is that an acute ruptured appendix superimposed itself in a patient who had recently been treated for cancer of the cervix.

C. S., colored, age 50 years. First seen on September 22, 1941, with an epidermoid carcinoma of the cervix, Schmidt 2. She was given 7,200 mgm. hours of radium without any x-ray. Her pelvic condition continued entirely satisfactory, but in March, 1943, she came in complaining of pain in her upper right quadrant. Since metastasis to the liver is a rather unusual occurrence in carcinoma of the cervix, it was felt that this was a gall-bladder infection and in no way associated with her cancer. An x-ray showed a non-functioning gall-bladder. She was explored on April 15, 1943, and a gall-bladder containing four stones was removed. An examination of her pelvis through the incision failed to show any evidence of involvement in the pelvis.

R. C. S., white, age 36 years. This case illustrates an opposite condition. This woman had a cancer of the cervix associated with an acute surgical condition which over-shadowed the malignancy. She first came in in December, 1933, because of irregular uterine bleeding and spotting. At the time she had an enlarged thyroid and a B. M.R. of plus 13 with some nervousness and some acceleration in her pulse rate. She was sent in the hospital with the request that a biopsy be made of an ulceration on her cervix but came back to the office two days later, very much disturbed because she said the gynecological service had ignored her pelvic condition and referred her to the surgical service for surgery. The biopsy proved to be squamous cell carcinoma and she was given a total of 5,835 mgm. hours of radium in two sittings. She was not given x-ray. In March, 1935, as she was showing no evidence of cervical malignancy, a thyroidectomy was done on her. She later married and was last seen in September, 1940, seven years after she was treated, with no evidence of malignancy.

The following case is quite interesting and I still am not sure of the pathological picture. B. P., colored, age 38 years. First came in in November, 1942. She had a squamous cell carcinoma of the cervical stump, Schmidt 2. In June, 1939, she presumably had had a supravaginal hysterectomy as she had not menstruated since that operation. On her hospital admission she had a four plus Wassermann. She was given 6,935 mgm. hours of radium at one sitting. We have a comment that she should have deep x-ray therapy but she lived out in the country, transportation was very difficult (after gas rationing went into effect) so it was all we could do to get her to the V. D. Clinic for treatment. In April, 1943, she reported for observation and stated that she was having bleeding from the rectum. The examination showed a large ulcer on the anterior rectal wall. There was no evidence of any involvement of the cervix and the indurated area did not involve the vaginal mucosa. We thought we were dealing with an entirely separate and distinct malignant lesion, probably an adeno-carcinoma of the bowel, and sent her in the hospital planning to do a colostomy on her and later remove the rectum and probably the cervical stump. A superficial biopsy was reported as showing no evidence of carcinoma, so the patient was anesthetized and a biopsy was taken through the entire thickness of the ulcerated lesion. Doctor Strauss reported that there was no evidence of malignancy but the sections were highly suspicious of gumma. He did not know the woman had a positive Wassermann. She was sent home and her anti-leutic treatment intensified. She was last seen on October 3, 1943. The rectal lesion was still present and she had developed a recto-vaginal fistula at the upper edge of the ulcerative lesion. I believe now that she would be better off if we had carried out our original intention and had done a colostomy and removed the rectum, even if we had operated for a syphilitic lesion.

Mrs. J. G., white, age 58 years. This case is a malignancy superimposed on an acute surgical condition. She had one pregnancy when she was 19 years old, had been delivered by Cesarean section and had had a supravaginal hysterectomy, so for forty years she had not had any vaginal bleeding. About two months before I first saw her she was admitted to the hospital with a fractured hip and, while convalescing from this, developed a gangrenous gall-bladder which was drained. Following that she had an acute myocardial failure, and a short time later began with vaginal bleeding. In July, 1941, she was given 5,300 mgm. hours of radium for a squamous cell carcinoma of the cervical stump, Schmidt 2 plus. Owing to her general condition, her age, and the fact that she had no pelvic organs, she was not given x-ray. In June, 1942, she was

returned to the hospital because of a recurrence of her carcinoma in the anterior vaginal wall and around the meatus. Deep x-ray was rather belatedly started on her but she tolerated it so poorly and became so nauseated it was discontinued. She died on November 15, 1942.

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DISCUSSION

DR. JOSEPH D. COLLINS, Portsmouth: I am sure that all of us have enjoyed Dr. Rawls' paper and the point which he has brought up is of utmost importance. The idea that a cancer victim is not entitled to treatment for intercurrent pathology is certainly a wrong one. Unfortunately the same notion prevails with regard to syphilis. Often various symptoms and diseases are ignored and allowed to go untreated just because the patient has a positive Wassermann or Kahn test. Dr. Rawls has rightly said that the word "cancer" carries a sinister significance, and to the average individual a diagnosis of cancer is equivalent to pronouncing a death sentence. This no doubt is due to the mistaken idea that the prognosis of cancer is always hopeless. How often do we hear the expression that when radium is used the patient always dies. Unfortunately the fatal cases are the ones which receive the most publicity. If it were possible for the public to be informed about the cured cancers I am sure a great deal of pessimism regarding cancer would be dissipated. Most individuals are very proud of their recovery from surgical operations and many delight in recounting the number of gall stones removed or the length of their appendix, but it is seldom indeed that one sees a patient who will even discuss his operation or any other procedure performed for the cure of his cancer. On the other hand, the patient who recovers from cancer usually keeps silent and will frequently say that his hospitalization was due to some other disease. This is no doubt due to the mistaken idea that the patient who has had cancer, although cured, is more or less disgraced. It is looked upon as a stigma which should not be discussed or referred to in his presence.

The cases which Dr. Rawls has cited are certainly most interesting and these patients are to be congratulated on his promptness in recognizing the conditions present and his competency in correcting them.

War-Time Graduate Medical Meetings

Are held under the auspices of the American Medical Association, the American College of Physicians, and the American College of Surgeons. Civilian as well as Service physicians are invited to attend and participate in the program.

The May meetings in Virginia are as follows:

FORT EUSTIS, VIRGINIA

May 11—Psychosomatic Medicine—Dr. Claude L. Neale.
May 25—Anesthesia—Selection and Contra-indications—Captain James P. Curran, M.C., U.S.A.

CAMP LEE, VIRGINIA

May 5—Rheumatism—Captain Joseph L. Hollander, M.C., U.S.A.
May 12—Respiratory Diseases and their Treatment by Chemotherapeutic Agents—Dr. Henry B. Mulholland.
May 19—Traumatic Lesions of the Abdomen—Dr. Carrington Williams.
May 26—Modern Diagnosis and Treatment of Pulmonary Tuberculosis—Dr. A. Barklie Coulter.
June 2—Aviation Medicine, General—Dr. Ludwig G. Lederer.

LANGLEY FIELD, VIRGINIA

May 2—Malaria (Clinical Manifestations and Therapy)—Dr. Carlton J. Casey.
May 9—Psychosomatic Medicine—Lt. Sidney U. Wenger, M.C., U.S.A.
May 16—Traumatic Arthritis—Lt. Commander Judson D. Wilson, M.C., U.S.N.R.
May 23—Rheumatism—Major Terence L. Tyson, M.C., U.S.A.
May 30—Neurocirculatory Compression of the Scalenus Anticus Muscle—Dr. Robert L. Payne.
June 6—Modern Diagnosis and Treatment of Pulmonary Tuberculosis—Dr. Dean B. Cole.
June 13—Respiratory Diseases and their Modern Treatment—Dr. Porter P. Vinson.
June 20—Treatment of Cardiovascular Emergencies—Dr. William B. Porter.

NORFOLK NAVAL HOSPITAL, PORTSMOUTH, VIRGINIA

May 11—Psychosomatic Medicine—Captain Charles A. Spangler, M.C., U.S.A.
May 25—Drainage of the Pleura with Particular Relation to Chest Injuries—Dr. I. A. Bigger.

RECENT ADVANCES IN THE PHARMACOLOGY OF NICOTINE*

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and
PAUL S. LARSON, PH.D.,
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Richmond.

During the past decade a number of investigations on the pharmacology of cigarette smoke have been made in our laboratory, and it is our wish to summarize here the results of those observations which might be of especial interest to physicians. Since nicotine is usually considered to be the most important constituent of cigarette smoke insofar as the systemic effects of smoking are concerned, this discussion will be largely limited to studies revolving about this one single component.

RESUMÉ OF PERTINENT GENERAL INFORMATION

Nicotine in the Cigarette and in the Cigarette Smoke.—The average American cigarette weighs one gram, is 70 mm. long, and 26.6 mm. in circumference. It contains a mixture of tobaccos, some domestic and some Turkish, so proportioned that the finished product contains about 2 per cent nicotine by weight. This corresponds to about 20 mg. of nicotine per cigarette. In addition, there is present a hygroscopic agent and flavorings characteristic of each brand. The paper consists of pure cellulose rendered to proper porosity by admixture with calcium carbonate. Contrary to popular belief no oxidizing substance such as potassium nitrate is added to the paper or tobacco.[1, 2]

Smoking habits vary widely between individuals. On an average, however, it may be assumed that the puff volume is about 35 cc. taken at minute intervals for a total of ten puffs per cigarette. When a cigarette is mechanically smoked under these conditions to a butt length of 23 mm., it is found that approximately 35 per cent of the nicotine in the tobacco smoked is completely destroyed at the burning tip; an equal amount goes off in what is called the "side stream" smoke which billows into the atmosphere from the burning tip; 6 per cent is collected by filtration into the butt, and 22 per cent is transferred into the "main stream" smoke.[3] It is this latter smoke with which we are primarily concerned, since this is the fraction which is taken into the mouth and, usually, subsequently inhaled. The 22 per cent carry-over of nicotine in the "main stream" smoke corresponds to about 3 mg. In other words, of the 20 mg. of nicotine present in the whole cigarette, 3 mg. are taken into the body by the average smoker.

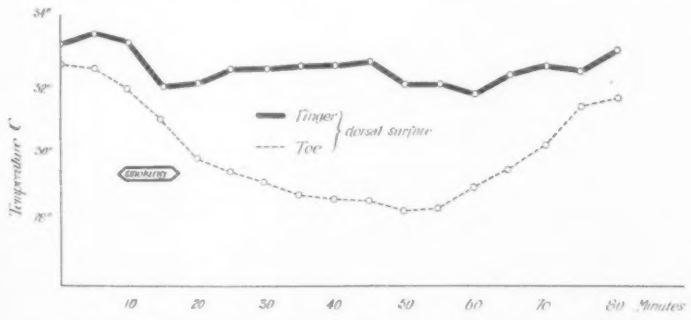

Fig. 1. Effect of cigarette smoking on the peripheral temperature of an habitual inhaling smoker. (7)

Absorption of Nicotine During Smoking.—The question naturally arises as to how much of this 3 mg. of nicotine in the main stream smoke is absorbed into the blood stream. Recent studies have shown that in the non-inhaler only a small fraction is retained, amounting to approximately 12 per cent.[4] On inhalation, however, most of the nicotine is absorbed by the respiratory mucous membrane, the actual values running as high as 98 per cent.[4, 5, 6]

*Read before the annual meeting of the Medical Society of Virginia, at Roanoke, October 25-27, 1943.

RESUMÉ OF EXPERIMENTS FROM THIS LABORATORY

Systemic Effects of Smoking.—Our investigations of the systemic effects of smoking have been limited chiefly to its effects on the circulatory system. Weatherby,[7] working in this laboratory, found, in keeping with other observers, that on inhalation of cigarette smoke subjects under basal conditions showed a temporary rise in systolic blood pressure of from 10 to 25 mm. Hg, an increase in pulse rate of from 5 to 20 beats per minute, and a depression of the skin temperature of the fingers and toes of from 2° to 7° C. Figure 1 illustrates the typical responses of the peripheral temperature to cigarette smoking which he obtained in habitual, inhaling smokers. Non-inhaling smokers showed only slight changes at the most. Weatherby ascribed these effects largely to the nicotine present in the smoke. Proof of this was obtained from experiments in which his subjects smoked *completely* denicotinized cigarettes with consequent marked reductions in the phenomena listed above. He found, furthermore, that on restoration of the nicotine to these denicotinized cigarettes their smoke produced the same results as originally obtained before denicotinization (fig. 2). Similar results on blood pressure and pulse rate indicative of the role of nicotine in provoking these circulatory responses in man were obtained here by Main[8] (fig. 3), and by us[9] in experiments with cigarettes made with Burley tobacco naturally low (0.13 per cent) in nicotine. This series of results are in accord with those of most other observers. However, one pair of investigators[10] offer evidence that nicotine is not the important agent in cigarette smoke so far as these effects on the circulatory system are concerned, and attribute these changes to a reflex mechanism activated by irritation of the respiratory mucous membrane. In light of the observations discussed above, we cannot agree with this concept which would exclude nicotine as a factor of major significance in regard to circulatory changes following smoking. Further evidence on the role of nicotine in this connection is to be had from experiments which we have made on animals in which we have shown (fig. 4) that the intravenous injection of solutions made by dissolving cigarette smoke in water leads to an increase in blood pressure entirely related to the nicotine pres-

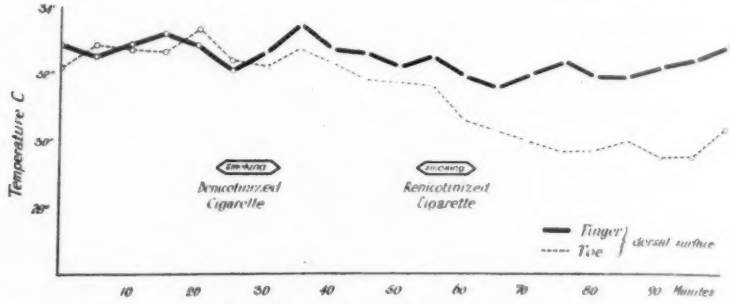

Fig. 2. Effect of smoking denicotinized and renicotinized cigarettes on the peripheral temperature of an habitual inhaling smoker. (7)

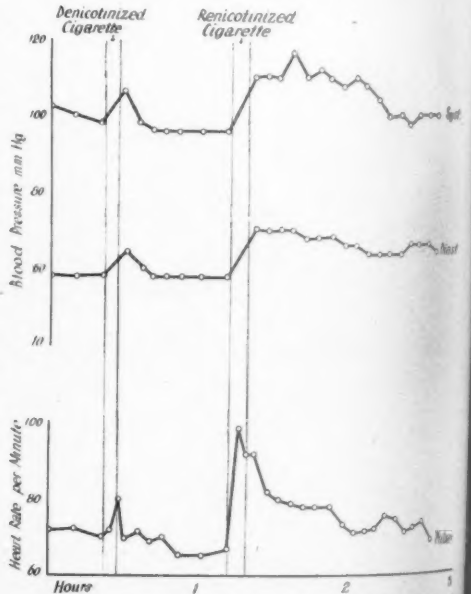

Fig. 3. Effect of smoking (exaggerated technic) denicotinized and renicotinized cigarettes on the blood pressure and pulse rate of an habitual inhaling smoker. (8)

ent.[11] Interestingly enough, by this method it has been possible to differentiate between various popular brands of cigarettes, chemical analyses of which demonstrated differences in nicotine content.[11]

Because of the clinical importance seemingly attached to the effect of smoking on peripheral temperature it appears worthy of note that Weatherby[9] found that various physiologic and psychic stimuli such as reading, talking, sudden noises, and the drinking of cold water evoked skin circulatory changes comparable to those produced by smoking (fig. 5). Somewhat similar findings were reported as long ago as 1909 by Bruce, Miller and Hooker.[12] Even more significant, from a practical point of view, was Weatherby's observation that cigarette smoking had no appreciable effect on peripheral temperature while the subject was up and active. From this it appears that what smoking does to a subject in a basal condition is not necessarily a true reflection of what it does when the subject is up and about. This distinction is deserving of further study in regards to other physiologic effects of smoking.

For some time now we have had in progress studies dealing with the effect of tobacco smoke on rats. In this experiment the animals are exposed, beginning at weaning age, to cigarette smoke daily under conditions closely simulating human smoking. As pertinent to the question of the permanent effect of cigarette smoke on blood pressure, it may be pointed out that these rats after eighteen months of treatment do not show blood pressure values different from those of the controls. These results are particularly striking when one considers that eighteen months represents one-half of the life span of a rat. However, while these rats show no chronic hypertensive effects from cigarette smoke, we have found that, in keeping with experiments on man, immediately after exposure to cigarette smoke, the blood pressure of these rats show some temporary elevation.

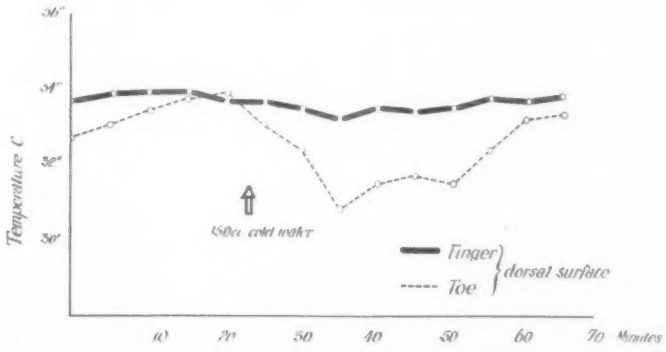

Fig. 4. Effect of drinking cold water on the peripheral temperature. (7)

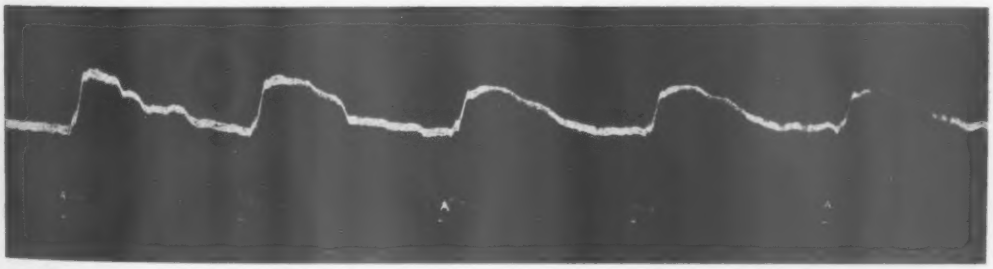

Fig. 5. Effect on blood pressure in a dog of the intravenous injection of nicotine acetate solution (0.1%) (N) and a cigarette smoke solution of the same nicotine content (A). Anesthesin, Dial: volume of solution injected, 0.03 cc. x Kg.; time, 6 seconds. (11)

Fate of Nicotine in the Body.—We have studied this problem both in the case of the human smoker

and in dogs after injection of nicotine. In man, we have found,[13] as have others,[14] that approximately 10 per cent of the nicotine calculated as having been absorbed was eliminated in the urine, the quantity, however, being governed by the number of cigarettes smoked, whether or not inhalation was practiced, and finally by the pH of the urine. Theoretical and practical considerations[15, 16, 17, 18, 19] would lead one to predict that less nicotine would be eliminated when the urine is maintained alkaline than when it is maintained acid. Actually, as seen from Table I, this expectation is fulfilled and only one-fourth as much nicotine was found in the urine when it was kept alkaline as when it was kept acid. We believe that this is due to resorption of the nicotine alkaloid by the mucous membrane of the urinary tract from an alkaline urine.[13]

TABLE I.

Subject	Cigarettes smoked daily	Amount nicotine retained from smoke (calculated)	pH of urine	Nicotine eliminated mg./24 hr.	Per cent urinary elimination of nicotine retained from smoke
1 (inhaler)	40	mgm. 115	5.5	12.7	
			5.0	17.3	
				av. 15.0	13.04
			7.1	2.1	
			7.6	3.2	
				av. 2.65	2.30
2 (inhaler)	18	52	5.6	5.37	
			5.7	6.27	
				av. 5.82	11.19
			7.1	2.09	
			7.2	1.30	
				av. 1.69	3.25
3 (non-inhaler)	40	14	4.9	0.24	
			5.2	3.00	
				av. 1.62	11.57

Effect of the hydrogen ion concentration of urine on the amount of nicotine eliminated by cigarette smokers. (13)

The fate of the nicotine not eliminated in the urine is unknown. Some traces are probably present in all secretions. However, the bulk of the nicotine retained during smoking must be either stored, which is unlikely, or chemically altered in the body. In experiments on dogs we have found,[20] as in man, that only about 10 per cent of administered nicotine is excreted unchanged in the urine. We can only conclude that the remainder (about 90 per cent) is chemically altered in the body. The pyrollidine component of the nicotine molecule would seemingly be the most likely site of chemical change since available information indicates that the pyridine molecule is not split in the animal body. With this in mind we have examined the possibility that the pyrollidine component of the nicotine molecule might be completely split off or reduced to a one carbon side chain. However, analysis of the urine of dogs to which nicotine had been administered subcutaneously in divided doses totaling 3 mg. per Kg. failed to reveal any increase above the basal level in their urinary excretion of nicotinic acid, nicotinuric acid, trigonelline or 1-methyl pyridinium hydroxide.

During the course of these experiments, we noted that there appears in the urine of dogs following administration of nicotine a compound that yields a red color when reacted with cyanogen bromide. This we have found is characteristic of nicotine derivatives in which the nitrogen of the group substituted in the *B* position of the pyridine molecule is unmethylated. Therefore, we have tentatively concluded that demethylation is a step in the detoxication of nicotine. However, this is not the only change that takes place. Simple demethylation of nicotine would yield nornicotine and we have shown that this substance is not present in urine following nicotine administration. Further work on this problem is in progress.

Regardless of the details of the mechanism by which the body rids itself of the absorbed nicotine,

available evidence indicates that this alkaloid is relatively quickly metabolized and eliminated. If this were not so it is hardly possible that one could smoke anywhere near the amount of tobacco one does without rather quickly developing major toxic phenomena. The exact rate with which man can detoxify and eliminate nicotine is not known; but rather definite information has been gathered on this point in our laboratory on rabbits. Weatherby[20] injected rabbits with fractions of the known fatal dose at regular intervals (Table 2), and found that their maximal

TABLE 2

Single dose, mg./kg	Dose interval, min.	No. animals in group	Total Administered mg./kg.	Total eliminated mg./kg.	Rate of elimination mg./kg./min.
1	1	20	8.3	2.1	.253
1	1½	20	12.0	5.8	.322
1	2	20	19.3	13.1	.339
1	3	10	23.7	17.5	.246
1	4	10	27.0	20.8	.193
1	5	10	31.6	25.4	.161

Rate of nicotine elimination by the rabbit following intravenous injection of 1 mg. per Kg. at different time intervals. (21)

capacity for detoxification and elimination was 0.339 mg. per Kg. per minute. Man is, in all probability, more susceptible to nicotine than the rabbit, and hence, if for no other reason, transference of these results directly to man is not justified. However, when regarded purely speculatively, these rabbits detoxified an amount of nicotine, when calculated on a weight to weight basis, equivalent to that which would be retained by man on inhalation of the smoke from eight cigarettes per minute.

The site of nicotine destruction in the body has been investigated by several experimenters.[22, 23] The consensus of these studies seems to be that the liver is the most important organ in the detoxification of nicotine. In at least partial substantiation of this we have found that mice in which liver damage had been produced by carbon tetrachloride tolerated repeated nicotine administrations poorly as compared to normal controls.

In conclusion, may we point out that in spite of the voluminous literature bearing on the physiological effects of tobacco smoke, the answers to many important questions still remain moot. This state of affairs obviously indicates the need for more extensive, more scientific, studies. We hope that we will be given the privilege of sharing in these and that from time to time you will allow us again to bring before you later reviews on the pharmacology of tobacco smoke and its constituents.

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DISCUSSION

DR. DOUGLAS G. CHAPMAN, Department of Medicine, M. C. V.: Dr. Haag and Dr. Larson have added much to our scientific knowledge of tobacco smoking. The opinions expressed about smoking are great in variability and low in reliability. The conflicting ideas are probably due to the individual's response to nicotine which varies from day to day.

Scientific facts obtained from human and animal experiments could only hope to be approximately correct. The effects of smoking are complex, our true knowledge is so slight and our opinions so divided that one's judgment may be more personal than scientific. First, some known facts: There is local irritation to the mouth, tongue, pharynx, larynx, and bronchi, and, if saliva is swallowed, the esophagus and stomach may also suffer. Cancer of the mouth, tongue, esophagus, larynx, and lungs is more common in smokers. Although smoking has increased in both sexes the percentage of cancer of these organs is decidedly greater among males. This fact is also true in countries where women have always smoked.

Respiratory System: Chronic cough and pharyngitis, with expectoration of thick mucus, is a common complaint in smokers. In Army discharges for tuberculosis, the smokers and non-smokers share alike.

Gastro-Intestinal Effect: Indigestion is our number one complaint in medicine. Tobacco, so far as I know, has never been prescribed as a cure for this complaint. It may decrease the appetite and inhibit hunger pains. It is credited with simulating most of the gastro-intestinal disorders. Women, again with an increase in smoking, have shown no increase in the prevalence of ulcer.

Secretion of Milk: Most reports show a slight to marked decrease in lactation and one report showed traces of nicotine in the milk after seven cigarettes were smoked in two hours. In several experiments no nicotine was found in the milk. In some countries where women have smoked since girlhood for generations there was no decrease in lactation and they raised and nursed large families.

Unborn Child: Some report an increase in fetal heart rate, while others report a slowing.

Reproductive System: It has been said that smoking tended to produce abortions but in harems where women spent most of their time smoking there were no abortions.

Growth: It is believed by some that tobacco may retard growth. Mice given large doses of nicotine over a long period of time outgrew the controls. More young people are smoking now than ever before—however, among the smokers and non-smokers their heights and weights are increased.

Blood Sugar is elevated with tobacco poisoning and in most heavy smokers. This is said by some to account for the pleasure of smoking. However, the smoker will not substitute a lump of sugar for a cigarette and he will tell you that his best smoke is after a meal and not on an empty stomach.

Allergy and Tobacco: The entire composition which enters into the manufacture of tobacco may play a role in allergy.

Physical Efficiency: John Adams, second President of the United States, after using tobacco for sixty years, complained that he found himself sickened by smoking only one-quarter of the amount of tobacco to which he had been accustomed. Mark Twain was asked in his latter years if he smoked. He said, "I do not smoke when I am eating and sleeping". In ordinary activities and a reasonable amount of smoking there may be no effect. It is likewise true that the highest muscular efficiency, endurance, skill, and ability to meet physical strain are incompatible with smoking.

Fatigue: Fatigue is marked in some individuals after smoking, and this varies from time to time in the same individual. I have frequently measured it in patients by having them climb twelve steps. They are generally below par and most of the weakness is manifest in the flexor muscles of the thigh. It is said smoking tires you when you are rested and rests you when you are tired.

Accumulated Effect: Tobacco headache is a good example of tolerance and intolerance in smoking. I believe this symptom, with those of fatigue and the circulatory system, offers the best method of investigating the cumulative effect of tobacco and accounts for the variations in symptoms.

Pain is increased by smoking in certain diseases, such as Buerger's disease, intermittent claudication, and in various forms of rheumatism, myositis, and fibrositis.

Circulation: Breathlessness on slight exertion, palpation, substernal distress, premature beats, precordial pain, changes in the T, QRS and P waves of the electrocardiogram are frequent findings in heavy smokers. Reports show that auricular fibrillation, auricular flutter, sino-

auricular heart block, auriculoventricular heart block are also occasionally precipitated by smoking. After coronary infarction by ligation, smoking markedly alters the electrocardiogram in the acute stage and the heart's tolerance to nicotine is one-fourth that of normal. There is less alteration in the sub-acute stage and still less in the chronic stage.

In one review of 15,000 smokers and non-smokers, there were ten per cent more than the average with organic heart disease.

We frequently hear: "Your legs or your cigarette", so one must decide for himself whether the pains or the pleasures of smoking are worth while.

Different parts of the same individual differ on sensitivity as individuals differ from each other. Each individual may change his state and condition from day to day and his reaction will vary accordingly. Nicotine is at first a stimulant to the sympathetic nervous system, then a depressant, and the state of the individual's nervous system may be the deciding factor in his or her reaction to smoking.

LT. COMMANDER EMIL BOGEN, M.C., U.S.N.R.: The pressor effect of nicotine, evidenced by rise in blood pressure, lowering in peripheral temperature, and visible changes in skin color, etc., is one of the most remarkable and consistent effects of this drug, and is of considerable pharmacological and clinical importance. The fact that such changes may be produced by other mechanisms, reflex or direct, or that they may be absent in other conditions, such as activity, should not be permitted to obscure this clearcut general phenomenon. The experiments reported by Haag and Larson show that the lowering in the temperature of the toe results from the nicotine in tobacco smoke, more than from all other factors combined. The relative effect of physical and chemical factors in the slight effects noted with their denicotinized cigarettes might incite further inquiry.

The studies of absorption and excretion of nicotine are interesting, but the results are not yet sufficiently extensive or convincing to resolve remaining doubts, or to explain just what does happen to the nicotine in the smoke. That some is absorbed and excreted unchanged seems evident, but just what happens to the remainder, though partially illuminated by this work, still requires further investigation.

The effect of nicotine on the peripheral circulation is important, but is not the only effect of this drug which may be of clinical importance, and so similar studies on the other manifestations of its action, similar to those here reported, are to be desired. Moreover, there are many effects of smoking which do not seem to be ascribable to the nicotine in the tobacco, and it is to be hoped that Dr. Haag and Dr. Larson will continue their studies and help to clarify them. The preparation of cigarettes *completely* denicotinized allows investigation of such effects which could not be made with the alleged denicotinized cigarettes on the market. It is to be hoped that such completely denicotinized cigarettes might also be made commercially available, so that physicians who have patients in whom nicotine is contraindicated, as in Buerger's disease, might make clinical trial of such products.

DR. HAAG, closing the discussion: In closing, I should like to thank Dr. Chapman for his excellent discussion and also to thank Dr. Bogen for his.

Dr. Chapman and Dr. Bogen have raised so many interesting and important points that I think it would require a great deal more time than is available this morning for us to go into these. I hope, however, that this will be possible at some early future date.

Thank you very much.

Physical Therapy Personnel Needed.

Realizing the acute need for physical therapy personnel, partly resulting from the war, The National Foundation for Infantile Paralysis has just made a two-year grant totaling $34,080 to the Stanford University School of Health (Women) at Stanford University, California.

This grant, which is in addition to other funds given by the National Foundation to this University, is for the two-fold purpose of strengthening the physical therapy technicians' school and of preparing syllabi and text materials for the use of physical therapy instructors and their students.

Under this program selected students will be provided specialized training designed to prepare them to become skilled teachers of physical therapy.

TOXICITY OF TRI-NITROTOLUENE*

ALEXANDER MCCAUSLAND, M.D.,†
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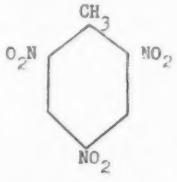
and
RICHARD F. HAWKINS, M.D.,
Radford, Virginia.

The toxicity of tri-nitrotoluene is somewhat better understood in World War II. This article is simply intended to present a review of the medical literature on the subject together with such information as has accumulated during the past two years in observing this type of industrial exposure. The section on pathology is taken entirely from the literature as there have been no fatal cases in the experience of the authors.

Chemistry of Manufacturing Process

Toluene



Tri-Nitrotoluene

T.N.T. is a pale, yellow, crystalline powder produced by completely nitrating Toluene with a mixed acid (sulphuric and nitric acid).

INCIDENCE OF T.N.T. POISONING IN WORLD WAR I

In England alone during 1916 there were 181 cases of T.N.T. jaundice; of these 52 deaths occurred.[1] In 1917 England reported 189 cases of T.N.T. jaundice, with 44 deaths. In Bavaria during the period of 1917 to 1918 there were 1,000 cases of toxic T.N.T. jaundice with 113 deaths. In the United States 7,000 cases of T.N.T. jaundice with 105 deaths were reported during a period covering 20 months. During the first 7½ months of 1917 the incidence of T.N.T. jaundice reached its peak with 17,000 cases of T.N.T. jaundice and 475 deaths.[2] During the last half of 1917 the incidence of T.N.T. poisoning decreased rapidly, although many workers were engaged in the manufacture of T.N.T. This sharp decline in the incidence of T.N.T. poisoning during the late months of the first world war was undoubtedly due to a better understanding of the toxicity, prevention, diagnosis and treatment of T.N.T. poisoning. These figures serve not only to prove that T.N.T. may be an etiological agent of a serious illness with a high mortality rate, but also that the morbidity and mortality can be significantly reduced by a better understanding of its toxicity, by proper preventive measures, and by early diagnosis and proper treatment in those cases of T.N.T. poisoning that occur in spite of our best preventive efforts.

CHANNELS OF ABSORPTION

1. Skin Absorption

The skin is the chief channel of entrance of T.N.T. into the body.[3] Skin absorption is increased in the presence of oily skin, sweaty hands in hot weather.[3] The area of exposed skin surface is reduced by the use of specially designed protective uniforms. These uniforms leave exposed only the hands, face, neck, and possibly the ankles. Gloves can further reduce the area of skin exposure if properly used. If T.N.T. is allowed to get inside of the gloves, the exposure of the skin of the hands is intensified, especially in warm weather when gloves cause increased sweating of the hands.

Skin varnishes have been used as a protective coating with the hope of preventing skin absorption, but have met with little success.[2] Skin cleanliness is the most effective means of reducing skin absorption. Skin absorption of T.N.T. is said[3] to be significantly less in women workers than men. This is explained[3] by the comparatively higher standard of cleanliness among women. A thorough shower and a change from work uniform to clean clothes at the end of the work shift will prevent further absorption, by removing the T.N.T. from the skin. It is to be pointed out, however, that a soap and water shower alone will not remove all of the T.N.T. from the skin. It has been shown that as much as a half of a gram of T.N.T. can be extracted from the 10 per cent sodium sulphite solution used on a

*Read before the Roanoke Academy of Medicine in May, 1942.

†Medical Director, Radford Ordnance Works, Radford, Va.

worker's hands as an after rinse following thorough soap and water scrubbing.[3] Sodium sulphite is one of the best solvents for T.N.T. and in a 10 per cent solution is harmless to the skin. Sodium sulphite solution turns red when it reacts with the T.N.T. on the skin and this color change impresses the worker as to its effectiveness, further qualifying the solution as an ideal after wash.

2. Alimentary Tract

The alimentary tract is also an important route of entry of T.N.T. into the body. The compound is quite soluble in saliva and gastric juices.[3] Smoking is not compatible with T.N.T. handling because of the fire hazard; therefore, chewing of tobacco becomes a habit with most workers. The T.N.T. laden hand of the worker placed into the tobacco package can start T.N.T. on its way into the intestinal tract. Likewise, the failure of the worker to wash his hands before eating his lunch leads to alimentary absorption of T.N.T.

T.N.T. poisoning has been produced experimentally by the gastro-intestinal route, in animals, by feeding the animal milk saturated with powdered T.N.T.[3]

3. Respiratory Tract

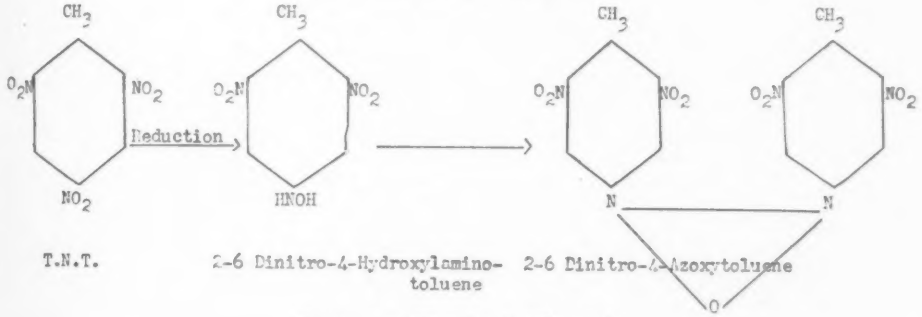
A less important route by which T.N.T. enters the body is the respiratory route. T.N.T. enters the body by way of the air passage in two forms, dust and vapor.

It has been estimated that a worker engaged in melting T.N.T. may absorb 16 milligrams of the material in an 8 hour shift by breathing its vapor.[3] It has been further estimated that a worker engaged in sweeping T.N.T. off of the floor may absorb as much as 9 milligrams through the air passage in a single 8 hour shift.[3]

FATE OF T.N.T. IN THE BODY

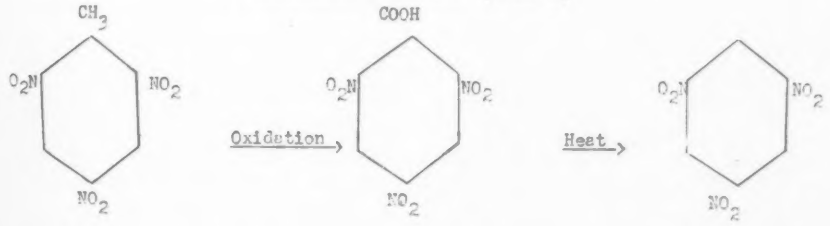
T.N.T. is said to undergo both reduction and oxidation within the human body.[4] The fate of T.N.T. in the body varies with different individual workers.

REDUCTION THEORY:[4] (Webster)



T.N.T. 2-6 Dinitro-4-Hydroxylamino-toluene 2-6 Dinitro-4-Azoxytoluene

OXIDATION THEORY:[4] (Watson)



T.N.T. 2-4-6-Tri-Nitrobenzoic Acid 2-4-6-Trinitrobenzene

The Webster test,[3] which is a qualitative color test for detecting T.N.T., is based on the fact that a solution of T.N.T. assumes a purple-red color when an alcoholic solution of potassium hydroxide is added. This test is negative when applied to the fresh urine of a T.N.T. worker, whom we know has absorbed large amounts of T.N.T.[3] However, if one modifies the Webster test by first acidifying the urine before the ether extraction, the extract so obtained gives the purplish-red color upon addition

of the alcoholic solution of potassium hydroxide. It has been suggested[3] that some derivative of T.N.T. giving the same color test as T.N.T. is being excreted in the urine of the T.N.T. worker. The only derivative of T.N.T. giving the same color test is the hydroxylaminotoluene derivative. Thus it has been suggested[3] that a part of the absorbed T.N.T. is broken down in the body into a hydroxylaminotoluene derivative which is excreted in urine.

It is suggested[3] that part of the T.N.T. is oxidized to trinitrobenzoic. Trinitrobenzoic is much less toxic than T.N.T. or its reduction product dinitrohydroxylaminotoluene.[3] This difference of toxicity has been explained[3] by the fact that trinitrobenzoic acid is extremely water soluble and is therefore rapidly removable from the body through the kidneys, whereas, T.N.T. and its hydroxylaminotoluene reduction product are poorly soluble in water, and are therefore eliminated from the body in the urine with difficulty.

The T..NT. and its reduction products retained in the body tissues are mobilized by the ingestion of alcohol.[9] T.N.T. workers frequently volunteer the information that even a small drink of whiskey or beer will cause them to feel warm all over, experience a tightness in the chest and a feeling of fulness, in the head, far beyond that normally experienced from the amount of alcohol taken. Many stories are told of T.N.T. workers passing out on barroom floors after the ingestion of only a small amount of alcohol.[9]

It has been suggested[3] that the difference in resistance shown by different workers to T.N.T. may be explained on the basis that the more resistant workers oxidize the methyl group of T.N.T. more readily, thus forming trinitrobenzoic acid which is rapidly excreted from the body through the kidneys, whereas the more susceptible workers reduce the T.N.T. to its hydroxylaminotoluene derivative which is eliminated from the body with much difficulty.

PATHOLOGY

The toxic action of T.N.T. is clearly shown at autopsy in the liver and the bone marrow. The most serious effect is the acute yellow atrophy of the liver.[8] In a patient dying from hepatitis following exposure to trinitrotoluene one may find the following:

1. Liver

Gross histological pictures are characteristic of the classical acute yellow atrophy.[8]

2. Spleen

The spleen is of normal or slightly increased size and shows some softening. Some observers consider moderate enlargement characteristic.[8] Microscopically, the blood sinuses are somewhat enlarged and contain large endothelial phagocytes filled with erythrocytes and hemosiderin.[8]

3. Kidney

Gross examination shows much bile staining and changes characteristic of acute toxic nephritis.[8]

4. Lungs

Grossly, the lungs are normal except for bile staining. A few instances of hemorrhagic infarction of the lungs have been reported.[8]

5. Adrenals

The tissue stroma shows changes similar to those described for the other reticulo-endothelial organs. Small areas of medullary cells are occasionally replaced by erythrocytes.

6. Bone Marrow

The bone marrow findings at autopsy have no one consistent pattern. The marrow varies in color from a pale pink with grayish areas to dark red. The red hyperplastic type of marrow is more frequently found. Turnbull's[3] microscopic examinations indicate a relative excess of erythroblastic activity and a decrease in the number of megalokaryocytes; numerous plasma cells and large phagocytes containing pyknotic nuclei, erythroblasts, erythrocytes and iron-containing pigment.

In the later stages of poisoning the aplastic picture may predominate with few or no myelocytes or nucleated red cells.[8]

7. Lymph Nodes

These contain numerous large mononuclear phagocytes loaded with granular hemosiderin.

8. Other Organs

Petechial hemorrhages of the pericardium, pleura and stomach mucosa have been reported.[8] The brain and other body tissues reveal little pathology aside from generalized icteric staining.

MANIFESTATIONS OF ANOXEMIA

Inasmuch as T.N.T. is fat soluble it is absorbed by the erythrocytes and acts to change the oxyhemoglobin into a mixture of methemoglobin and nitric oxide hemoglobin.[9] Neither methemoglobin nor

nitric oxide hemoglobin can act as a satisfactory carrier of oxygen and thus an oxygen deficiency results. This oxygen deficiency gives rise to numerous manifestations in the worker.

Cyanosis typically appears on the lips, fingernails, tongue, and mucous membranes of the affected worker. This cyanosis is characterized by a "greyish mauve" shade and may therefore be differentiated from the cyanosis of cardiac or lung disease.[6] Artificial oxygen inhalation has little effect on the cyanosis but does benefit the patient by slowing the pulse and decreasing the respiratory rate.

Dyspnea on exertion occurs as a manifestation of anoxemia.

Many T.N.T. workers complain of dizziness, especially when bending over. This dizziness has been explained[6] as due to temporary functional abnormality of the cerebellar centers incident to the cerebellar anoxemia.

A complaint of the T.N.T. workers is muscle cramps. This symptom is often hard to evaluate in workers doing heavy manual labor, but in cases where the muscle cramps are due to T.N.T. poisoning the explanation is probably based on an anoxemia of the muscles. This anoxemia is proportional to the amount of muscle exertion.

Headache is a frequent complaint and probably has its explanation in anoxemia of the brain.

T.N.T. Facies

The T.N.T. facies is characterized by a pallor comparable to that of shock with a grey-blue or slate like coloration of the lips, and of the lobes and helix of the ears.[6] The sites mentioned are selective. In the typical T.N.T. facies, it has been reported[6] that coloration is brighter than that seen in cyanosis of other types. The lips are a "lilac shade" in true T.N.T. facies, as compared to "ashen grey" lips of cyanosis of heart failure, pneumonia, or others. The color of the lips in T.N.T. intoxication is so unreal as to convey the impression, in women, that the wrong shade of lipstick has been used. The color is greatly modified by excitement or exercise on the part of the worker, such as rushing to get to work on time.[6] Artificial light also changes, surprisingly, the color of the T.N.T. facies. Under a mellow yellow light the color disappears and reappears when the worker steps into daylight.[6] A theory proposed by Roberts[6] contends that T.N.T. stimulates the vasoconstrictor center, causing a local capillary stasis and thus an increased venosity of the blood in that area. This theory seems to be a logical explanation of the color of the lips and ears since the characteristic appearance is known to occur without other signs or symptoms of anoxemia, such as fatigue, and breathlessness. Furthermore, such facies may exist when the blood count is normal. In view of these observations it seems we should recognize the T.N.T. facies as a characteristic and distinct manifestation of T.N.T. poisoning.

Gastro-Intestinal Manifestations

An unpleasant taste is an almost constant complaint of workers during the period of contact with trinitrotoluene.

Epigastric pains often seem to be just hunger pains resulting from stimulation of the appetite by T.N.T. Other instances are undoubtedly due to a T.N.T. toxic gastritis.[4] In some cases it is possible that already existing functional abdominal complaints are aggravated by T.N.T. Abdominal complaints are less frequent in men who come to work with a full stomach.

Nausea and vomiting may be due to a toxic gastritis resulting from the ingestion of T.N.T.[4] The only physical finding may be muscular tenderness resulting from the effort of vomiting. Vomiting may become so severe that intravenous salt replacement is needed.

Skin Manifestations

A characteristic yellow-orange staining may be found on the palms of the hands and soles of the feet of exposed men. This is not necessarily accompanied by signs of intoxication.

The small sharp crystals of T.N.T. may, by mechanical trauma, produce a dermatitis on those exposed areas of skin with which it comes in contact. Friction from the clothing aids in producing the irritation.

Another type of skin reaction appears to be true sensitization dermatitis. The patch test is helpful in making the diagnosis in such instances. One method of testing is to apply a 5 per cent alcoholic solution of T.N.T. to the skin and examine at 24, 36 and 48 hour intervals after application. Many workers never develop a T.N.T. dermatitis even though they are exposed for years. Some workers will develop a mild dermatitis early in their work which disappears subsequently even with continued exposure. Those who fall in this group seemingly

acquire an immunity or become desensitized from the first attack. Therefore, it is not necessary to remove a worker from the exposure on the basis of early, mild dermatitis. Other workers stand the exposure for months without a dermatitis and then suddenly develop the dermatitis. It would seem that these workers acquire a sensitivity or susceptibility from the exposure which they did not possess at the time they began the exposure.

We have no certain way of predicting just which worker will show susceptibility to T.N.T. in the form of a dermatitis, neither do we know which worker will be immune or develop immunity after a single attack. So beyond elimination of those candidates for exposure who give a definite history of frequent skin trouble, one proceeds largely by trial and error.

T.N.T. JAUNDICE

In experience reported[3] from World War I it has been estimated that 0.2 per cent of all workers exposed to T.N.T. developed toxic jaundice and that there was a 30 per cent mortality in this group. T.N.T. jaundice is usually, not always, a late sign of T.N.T. poisoning, but it is indeed a severe sign. Most workers who develop jaundice do so during the first six months of contact with T.N.T.

Every case of jaundice does not mean that actual parenchymatous liver damage is present.[5] Jaundice occurring in the absence of actual liver damage has been explained[3] on the basis of a phagocytic anemia with the deposition of hemosiderin and bilirubin throughout the body, because of the inability of the liver to excrete the pigment as fast as it is liberated from the erythrocytes.

It has been suggested[3] too that the icterus may in some cases be due to an increased viscosity of the bile leading to obstruction of the smaller bile ducts, without definite liver damage.

In other cases the icterus is due to extensive necrosis and atrophy of the liver, characterized as acute yellow atrophy. In jaundice due to liver damage, it is believed[3] that certain pre-existing pathological conditions affecting the functional capacity of the liver (alcoholism, cirrhosis, syphilis) may predispose the worker to a toxic jaundice. In the presence of this co-existing pathology it is possible that the action of T.N.T., or its reduction products, may result in greater damage to the liver cells than occurs in a person with a normal liver, or that such a person will have a smaller reserve of good liver tissue to risk against further hepatotoxic chemicals. This is a foundation for eliminating candidates for this exposure, who at the time of employment give a history of chronic alcoholism, syphilis, jaundice or liver disease.

Although T.N.T. jaundice usually follows or is associated with less serious symptoms, such as nausea, vomiting, cyanosis, etc., it may occur suddenly and in complete absence of any other signs of poisoning. The icterus in early stages is a characteristic lemon yellow color, rather than the greenish brown color of obstruction jaundice.

It is important that all physicians, the specialist and practitioner alike, in the neighborhood of T.N.T. plants, acquaint themselves with the manifestations that T.N.T. may cause. Inquiry into the patient's working conditions often gives a clue to the diagnosis. Correct early diagnosis is important if the affected worker is to have the best chance for recovery.

BLOOD CHANGES IN T.N.T. POISONING

Blood taken by venopuncture from a patient with severe T.N.T. poisoning is dark bluish red in color and has an increased viscosity. These changes in the gross appearance of the blood are thought to be due to a mixture of methemoglobin and nitric oxide hemoglobin.[7]

In patients with T.N.T. poisoning the erythrocytes may be small and fragmented. All workers who show marked changes in the blood have definite symptoms.[7] While one cannot rely on the character of the erythrocytes alone to indicate the degree of poisoning, it is possible to get a rough index as to the degree of poisoning in some instances from the hematologic examination.[7]

The anemia of early T.N.T. poisoning has been thought to be due to an increased rate of destruction of erythrocytes and not due to the toxic action of T.N.T. on the bone marrow.[7] T.N.T. is assumed to be absorbed by the cells and to change part of the oxyhemoglobin into a mixture of methemoglobin and nitric oxide hemoglobin. Disintegration of the erythrocyte follows. The red blood cell fragments are engulfed by endothelial phagocytes of the spleen, bone marrow, lymph glands and Kupffer cells of the liver where the hemoglobin is broken down into bile pigment and hemosiderin.[8] Although there is destruction of erythrocytes, no anemia

results so long as regeneration compensates for the destruction.[7] When destruction exceeds regeneration, anemia results.

Aplastic anemia is a late and rare complication of T.N.T. poisoning. In the latter stages of the anemia of a neglected worker the function of the bone marrow may be seriously depressed[7] due to an oxygen deficiency or other metabolic abnormality.

The aplastic anemia is almost always fatal and occurs in those workers who have been subjected to constant exposure, even in the face of anemia. Weekly blood examinations will detect the early preaplastic stages and thus prevent the anemia from progressing to the aplastic stage. Aplastic anemia may or may not be preceded by jaundice.[7]

In the early stages of T.N.T. poisoning there is often a polynuclear leukocytosis.[7] This leukocytosis is presumably due to stimulation of the haemopoetic system by T.N.T.[7] This initial leukocytosis is followed by a decrease in the leukocytes with an absolute increase in lymphocytes. Still later a leukopenia may develop with a diminished polymorphononuclear leukocyte count and a relative increase in lymphocytes. With aplasia of the bone marrow in severe T.N.T. poisoning, there is decreased output of polynuclear cells from the bone marrow, but at the same time the lymphocyte forming tissue may be unaffected.[8]

Eosinophilia in the absence of other cause can be looked upon as due to the effect of T.N.T.[7] Eosinophilia suggests good rather than poor marrow activity. Eosinophils are sometimes increased in T.N.T. dermatitis. The precise cause of the eosiniphilia is unknown, but may be an indication of an allergy to the chemical.

TREATMENT OF T.N.T. JAUNDICE

It is advisable that all cases of T.N.T. jaundice, even the very mild, be sent to the hospital as soon as they are detected. Hospital facilities are necessary for the indicated strict observation and vigorous treatment which may be needed. As soon as practicable after admission to the hospital the patient should have a thorough bath with warm soap and water, followed with an adequate after rinse of 10 per cent sodium sulphate, to remove any T.N.T. remaining on the skin. The patient should receive at least 20 grams of intravenous glucose daily.[5] The glucose serves to fortify and protect the liver, and since the liver may already be injured due to the toxicity of T.N.T., the protective value of glucose is an essential consideration. The diet from the beginning, in the absence of nausea and vomiting, and as soon as tolerated in such cases as are associated with nausea and vomiting, should be one high in carbohydrate and low in fat. The high carbohydrate content of the diet serves to fortify the liver.

The rational of ascorbic acid therapy is supported by animal experiments[4] which demonstrates that carnivorous animals are highly susceptible to T.N.T., whereas herbivorous animals are highly resistant to T.N.T. Carnivorous animals derive very small amounts of vitamin C from their diet, whereas herbivorous animals derive comparatively large amounts of vitamin C from their diet.

It would seem there is logical basis for the use of vitamin C therapy, both as a preventive and in the actual treatment of T.N.T. poisoning. In any event it can do no harm, since surplus vitamin C is excreted without evidence of harmful effects.

It has not been demonstrated that workers receiving excess vitamin C intake are less susceptible to the toxic action of T.N.T., than are workers receiving a normal intake of that and other vitamins. The worker with T.N.T. poisoning that has progressed to the stage of jaundice is likely to have a vitamin C deficiency due to the associated loss of appetite, nausea and vomiting; therefore, it is our feeling that vitamin C is of indicative value in the actual treatment of these cases.

Vitamin C, when given preventively, should not be given with the idea that it is a specific preventive, but rather for the purpose of supplementing the vitamin C content of the diet of the worker, thus taking care of any vitamin C deficiency that may exist in the diet of the worker. Controlled studies are needed to answer this question.

Liver extract in adequate dosage is advisable in jaundiced patients who show even the slightest anemia. Some patients may have an aplastic blood picture, yet the bone marrow be hyperplastic.[5] It has been suggested[5] that the aplastic anemia is due to the inability of the damaged liver to supply some factor essential to hemopoiesis. Furthermore, treatment of the anemia with iron alone will result, in many cases, in an adequate hemoglobin response, but a poor or delayed erythrocyte response.

Such drugs as the sulfonamides, chloral hydrate and amidopyrine should not be given to the patient

with T.N.T. jaundice. These drugs are capable of causing damage to the liver and bone marrow and may increase the damage to these organs caused by T.N.T.

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Pharmacy and Medicine Working Together.

Harvey B. Hagg, M.D., head of the Department of Pharmacology, Medical College of Virginia, says that one of the most difficult problems in the teaching of medical students is that of prescription writing. Perhaps no single facet of medical instruction is so frequently the butt of adverse criticism, not only from physicians themselves but from those responsible for compounding prescriptions, namely, pharmacists. Those who teach pharmacology to medical students are well aware of this situation, and many have been the methods used in attempting a solution. In this connection it may be of interest to outline briefly a plan which is being tried at the Medical College of Virginia.

A prescription is a message from a physician to a pharmacist, and so the better these two understand one another the more smoothly will this message be interpreted and executed. With this in mind the school of pharmacy here at the college was asked some several years ago to aid in the teaching of prescription writing to our medical students. This cooperation was gladly given. At present during the regular pharmacology course eight lectures are given by Dr. T. D. Rowe and Dr. Karl Kaufman, in which the more common pitfalls and errors of prescription writing, as the pharmacist sees them, are brought to the attention of the medical students. In addition, other problems of mutual concern, such as, for instance, the intricacies of the narcotic laws, are discussed. While this program has now been in progress too short a time for final evaluation, it is already evident that this addition to the teaching of medical pharmacology is bearing fruit, and it is planned to continue this cooperative scheme on a more elaborate scale in the coming years. Fundamentally this would seem to be a sensible and logical procedure and for us here at the college, because of our unique organization, an especially feasible plan. (*The Virginia Pharmacist*, 28: 1944, 38.)

REPORTS AND OBSERVATION ON SOME THREE HUNDRED CASES OF WHOOPING COUGH*

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and
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It has been quite a few years since Petersburg had even a mild epidemic of whooping cough, and so we welcomed the opportunity to try, in a rather feeble sort of way, to evaluate the virtues of the modern methods of the prevention and treatment of this disease.

As you well know, pertussis ranks as one of the great causes of infant and childhood mortality. Although the medical profession still, in a large majority of cases, takes the attitude of "laissez faire", some even going so far as to say that the disease has to run its course and there is no necessity for bothering about prevention and treatment in whooping cough, the morbidity and mortality rates do not bear out this indifferent attitude as is shown by the fact that there were 2,405 cases reported to The State Health Department in 1942 with a mortality of 5 per cent, and so far in 1943 there have been 799 cases reported with a mortality rate of about 10 per cent.

Dr. White and I have diagnosed and treated 250 to 300 cases of whooping cough this year (1943). The statistical data of 200, on which records were kept, are as follows:

Males	80
Females	120
White	180
Colored	20
Mild	106
Moderately severe	60
Severe	34
Under 1 year of age	30
1-2 years of age	11
2-4 years of age	24
Over 4 years of age	135
Preventive inoculations	11
No prophylactic treatment	189

Of these 11 cases—

1 severe
3 moderately severe
7 mild

*Read before the annual meeting of the Medical Society of Virginia, at Roanoke, October 25-27, 1943.

COMPLICATIONS

Only 14 cases had complications
Only 2 deaths

One death was an infant three months old with severe whooping cough and was seen only when in extremis. The other, five months old, was cared for very poorly, had pneumonia at onset, from which it recovered, but the whooping cough became worse and the baby died.

TREATMENT

Our treatment of these cases during this epidemic consisted of—

1. Ascorbic acid
2. Elixir of gold chloride (Elixir BroMuriate)
3. Vaccines (antigens and nasal treatment with pertussis topogen)
4. Diatussin
5. Pertussin and the expectorant cough syrups
6. Sedatives, as indicated
7. Sulfa drugs.

Dr. White thinks very highly of ascorbic acid in large doses. I prefer vaccines. Both use elixir of gold chloride but I am partial to Diatussin. We both used sulfadiazine in cases of pneumonia and bronchitis complicating the disease, and some uncomplicated cases seemed to respond nicely to sulfathiazole.

The mortality was only 1 per cent, both deaths in infants of a few months, under poor environmental conditions.

As only 11 children had the disease who had previously been immunized; and as we cannot report on the number of cases who were exposed to the disease after having been immunized, we can only suggest from this study that the prophylactic treatment is of benefit. I think it is fair to say, however, that both Dr. White and I immunize our babies routinely and both have immunized many hundreds since its recommendation by the health authorities and the American Academy of Pediatrics.

As only 11 cases, or about 5 per cent of this study, had received the prophylactic treatment, we think it follows that the measure is of value and should be continued and extended.

In conclusion, may I state that in my opinion, while we have no specific treatment that is of value in every case of whooping cough, each case still should be treated symptomatically and at times vigorously, as we have outlined above, or as it seems indicated to the individual physician. Many cases of severe whooping cough can ge greatly aided and complications avoided if the physician sees the case at regular intervals, outlines measures for its control, and prescribes remedies for its varying symptoms, thus bringing to a happy conclusion a disease which could easily be fatal by neglect and indifference.

DISCUSSION

DR. J. B. STONE, Richmond: Dr. McIlwaine's paper, I think, has very properly emphasized three important facts regarding whooping cough. The first is the seriousness of this disease in infants and small children; the second, the effectiveness of immunization; and, third, the relative inadequacy of the treatment often given, or of the agents available for treatment.

In regard to its seriousness, whooping cough is a disease so widespread that the lay public, and many doctors, are inclined to look upon it as a trivial disease. As Dr. McIlwaine has pointed out, it is not a minor disease. The mortality in infants under one year of age is twenty-five per cent; during the second year, it is ten per cent; for all ages, over two per cent. With such a high mortality, it cannot be looked upon as a trivial disorder.

With regard to the efficiency of treatment Dr. McIlwaine's figures, I think, are significant. In other series of cases, on a larger scale, other investigators have given similar results. For instance, in a report by Silverthorne of 747 cases immunized, there was an incidence of only 2.2 per cent, whereas in a control series with somewhat comparable numbers the incidence was 85 per cent. In another series, reported by Eldering and Kendrick, of 1,851 cases immunized there was an incidence of 2.8 per cent, whereas in the control series there was an incidence of 80.2 per cent. It is the experience of other workers that proper immunization with fresh, properly prepared vaccine is very effective in lowering the incidence of pertussis. It must be kept in mind, too, that to be effective the vaccine must be administered before the child is exposed to whooping cough. A period of at least three months following inocculation is necessary for full immunization to occur. Many people think it not effective because a child exposed to whooping cough a month or six weeks after inocculation develops the disease. If the vaccine is administered to young children and infants before an epidemic occurs it gives time for immunization to become effective.

If immunization is done at seven months of age it is much more effective than when the vaccine is administered at three months. Yet it is in the earlier ages that the mortality is so great. So we are between the devil and the deep blue sea. It is my own practice to give the immunization at five or six months of age and then give a stimulating dose later. There is a tendency for the immunity to decline over a period of time, and by giving a stimulating dose the immunity can be brought back. That should be kept in mind.

With regard to the treatment of the active case, I think Dr. McIlwaine is to be congratulated when we consider his mortality of only one per cent. When compared to the general mortality of twenty-five per cent in that age group he indeed merits congratulation.

As I said in the beginning, specific agents for the active treatment of whooping cough are very meagre. The sulfonamides are coming into use for treatment of complications. Aside from these, other effective agents are rare. Two things used are hyperimmune rabbit serum and hyperimmune human serum. My own experience with rabbit serum has been very limited, but I have been favorably impressed. I have used rabbit serum in only two cases. One problem is the very high cost. It costs fifteen dollars a dose.

Where the hyperimmune human serum has been used in infants under two months of age the mortality was two per cent. The difficulty here is that the serum is not generally available to us in general practice. But these two agents do stand out as offering some hope.

As Dr. McIlwaine said, the wide variety of drugs used in treating whooping cough is an indication of their lack of efficacy. About all we can do in most cases is to try to prevent complications and endeavor to keep up the child's general nutrition. It is, of course, a very difficult procedure to handle these young infants when they actually have whooping cough. That takes us back to the question of the importance of routine immunization of all infants during the first year of life. Around the middle of the first year I think is generally considered the optimum time for immunization.

A STUDY OF THE VALUE OF A PERTUSSIS VACCINE*

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and
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In 1939, Kendrick and Eldering[1] published a report of the results obtained by using a pertussis vaccine washed once with saline, on a group of 1,815 children from eight months to six years of age. These children and the 2,397 controls were selected from a metropolitan area with an unusually stable population, 60 per cent of the families being home owners.

The results were based on a period of observation of thirty-nine months. Fifty-two cases of pertussis developed among the vaccinated children and 348 cases among the controls. The attacks among the vaccinated lot were less severe than among the controls.

Through the courtesy of the Bureau of Laboratories, Michigan Department of Health, a supply of this vaccine was furnished for use in the Child Welfare Clinic of the Pediatric Department at the Medical College of Virginia.

The vaccine consisted of a seventy-two hour growth of H. pertussis on Bordet-Gengou medium, washed off in a small amount of saline; the suspension then being filtered through cotton and centrifuged. The organisms were resuspended in saline with a killing agent and stored in a cold room until sterile. The killed suspension was standardized to ten billion organisms per cc. It was tested for agglutinability with specific antiserum and for ability to produce agglutinin in rabbits.

This vaccine was administered in the clinic by the nurse in charge under the supervision of the head of the pediatric department. It was given in four weekly subcutaneous injections of 1, 1.5, 1.5, and 3 cc., the last injection being divided into equal bilateral injections.

The cases were selected from children between the ages of eight months and four years who had not been previously exposed to pertussis nor vaccinated against it. However, this group also contained seven infants of approximately six months, and three children of seven years. The average age of the group was thirteen months and the median age was ten months.

The controls were selected from the same age group and within the same families in as far as possible. The large majority came from poor income or relief brackets and lived in crowded city areas.

The experiment ran from May, 1940, to September, 1943—or a total of thirty-nine months. In all, 317 children (75 white and 242 Negro) were vaccinated. One hundred thirty-two children (16 white and 117 Negro) served as controls. Of the vaccinated group, 300 received all four injections, 7 received three, 1 received two, and the remaining 9 received only one injection.

The cases and controls received a home visit by a case worker or nurse approximately every four months, during the thirty-nine month period of observation. The code for reporting exposure to the disease and severity of the disease when it developed was the same as that used by Kendrick and Eldering.[1]

VACCINATED GROUP. Among the 317 vaccinated children, a history of definite exposure was obtained in 34, indefinite exposure in 52. A number from both groups had repeated neighborhood exposures during this period. The home exposures were most intimate, patients and exposees often sleeping in the same bed and as one grandmother said: "even licking off the same sucker".

Thirty-six children did not develop the disease during varying periods following inoculations, but were lost before the end of the observation period, either by moving from the city or by moving within the city without leaving a forwarding address.

In the 291 cases followed throughout the entire period, 7 cases of pertussis developed, 1 of which

*Thanks are due Dr. Lee E. Sutton, Jr., and Dr. A. J. Borowski for reading the manuscript and for advice.
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Vaccine was furnished by Dr. Pearl Kendrick, of the Michigan Department of Health Laboratories.

could not be verified. The following table shows the pertinent facts concerning these cases:

Cases 1-2-4-5 must be thrown out of the series since no time was allowed for the development of active immunity before exposure.

Case 3 was said to have developed the disease about seventeen months after immunization by a second injection which continued about two months without any additional findings. One child, who is not included in the series, developed a fever after each of two injections and was not continued. There was an occasional complaint of a little local soreness or lump at the site of injection for two or three days.

NAME	AGE AT TIME OF INOCULALTION	NO. OF INJECTION	DATE OF EXPOSURE	LENGTH OF TIME AFTER INOCULATION BEFORE SYMPTOMS DEVELOPED	NATURE OF SYMPTOMS
1. J. L.	7 months	4	Before last inoculation	After last inoculation	Light
2. R. H.	18 months	1	After first inoculation	After first inoculation	Unknown Case not followed
3. R. J.	7 months	4	None determined	17 months	Mother vague. No confirmation at time of home visit.
4. B. B.	11 months	4	Before last inoculation	After last inoculation	Light
5. N. C.	9 months	4	Before last inoculation	After last inoculation	Moderate to severe
6. A. T.	8 months	4	None determined	20 months	Light
7. L. W.	12 months	4	None determined	3 months	Moderate to severe

mother who was unwilling to impart information and who would not state how long or how severely sick the patient was. The child was presumably well at the time of the home visit.

There remain two cases of verified pertussis, numbers 6-7. The severe case occurred in a 15 month old child three months after completion of immunizations. No history of exposure could be elicited. The mild case developed in a 28 month old child, twenty months after inoculation. No history of exposure was elicited. In addition, 3 of the above children developed cold and cough lasting approximately one week, after exposure to pertussis.

CONTROL GROUP. Among the 132 controls, a history of definite exposure was obtained in 3 cases, and of indefinite exposure in 23. From these, there developed 1 light, 1 moderate, and 2 suspicious cases of pertussis. Among those who gave no history of exposure there developed 23 moderate and 7 severe cases, making a total of 34 cases.

VACCINE REACTIONS. Three children developed a mild cold with cough, within a week after the last injection, which was considered a possible vaccine reaction. One child developed a fever after the

CONCLUSION

Because of unavoidable circumstances, there was a shifting and shortage of personnel on several occasions so that the project could not be followed through with the detail intended. However, it seems safe to draw the conclusion that the vaccine used is of definite value in preventing or minimizing the development of pertussis.

The results are presumbably even better than the figures show, since indications are that the parents' histories of exposures were inaccurate. It is safe to assume that many more children were exposed than are recorded.

The type of pertussis which developed in the controls indicates that the epidemiology and symptomatology for the years included in the study were average.

SUMMARY

A group of children given pertussis vaccine developed good protection against pertussis.

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AN OUTBREAK OF FOOD POISONING DUE TO STAPHYLOCOCCUS AUREUS

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Outbreaks of food poisoning due to bacteria or their toxic products are by no means rare in our experience. Each year a number of such episodes come to our attention. These usually involve small family groups and their occurrence is a matter of individual rather than of public sanitation. It has been our experience that these minor epidemics are reported to us only after the lapse of several days and examination of the foods concerned in the outbreak has been impossible. The patients are on their way to recovery and show no organisms in stool cultures.

On July 7, 1943, there were reported to us several cases of food poisoning, evidently attributable to a luncheon given twenty hours previously. Investigations in the field as well as in the laboratory gave results which enabled us to trace the outbreak in a satisfactory manner to its original source. The chronological order of events is followed in this narrative report.

About the first of July, a local caterer, called Mrs. A. for purposes of this report, received requests to serve food at two gatherings; one on July 5, food being served to about 45 guests at an afternoon wedding reception; the second on July 6, a luncheon for 70 ladies, wives of Service Officers stationed in Charlottesville. In anticipation of these two events, the caterer purchased fresh chickens which were boiled on the morning of July 5. The meat was picked over by Mrs. X, one of the three individuals handling this food. The portion designed for the wedding luncheon was made into a salad by the addition of home-made boiled dressing. This was served at about 4:00 P. M., five to six hours after its preparation. None of the 45 guests was made ill. A portion of the chicken salad was put aside in the refrigerator by the maid employed at the home where the wedding reception was held. This remained in the refrigerator for 48 hours. It was then eaten by the maid. She became ill three hours later with nausea, vomiting, abdominal cramps and diarrhea.

Chicken from the same source was kept in a mechanical refrigerator from the morning of July 5 until late in the evening. It was then made into jellied chicken by the addition of the concentrated chicken broth, the entire process being finished about 1:30 A. M. on July 6. It was kept in the mechanical refrigerator until about noon on July 6 and was served at the luncheon at 1 P. M. The menu served on that occasion consisted of a cold plate with jellied chicken containing peas, slices of hard-boiled egg with salad dressing; rolls, butter, cottage cheese and tomato salad, iced tea, and apple butter tart.

Many of those partaking of this food were made ill in from three to 42 hours. The median incubation period was five hours and the mean period 6.3 hours. The symptoms varied in intensity, but in general were characterized by nausea and vomiting, severe abdominal cramps, diarrhea and weakness.

Of the 70 persons partaking of this luncheon, we were able to acquire information from 61 by questionnaire and personal interviews by members of the Joint Health Department's nursing staff.

CLINICAL DATA

Of the 61 guests interviewed, 43 or 70 per cent were ill in some degree. Of 53 guests reporting that they ate some of every food at the banquet, 37 were made ill and 16 reported no illness. A few of these later, however, admitted slight abdominal discomfort. Of the eight women who chose only certain items of the menu, two were not affected. One of these ate everything but the cottage cheese; the other ate nothing but a sliced tomato. Six guests who ate only certain items on the menu were among those made sick. An analysis of their selections showed nothing significant, except that the only article of food eaten by all was the jellied chicken and peas.

An analysis of the symptoms suffered by the 43 sick individuals showed the following in decreasing order of frequency:

(1) Abdominal pain and cramps.
(2) Nausea.
(3) Diarrhea.

(4) Vomiting.
(5) Weakness.
(6) Dizziness.
(7) Generalized muscular aches.
(8) Headache.
(9) Prostration.
(10) Fever.

Most of the victims complained of multiple symptoms; for example, abdominal pain and nausea, each reported by 35 persons, coincided in 29 of them, or 83 per cent. Two individuals who were sick did not report symptoms in detail.

Forty-one individuals reported with accuracy the duration of their illness. The maximum was 48 hours, in seven instances; the minimum two hours, in a single individual. The mean duration of illness was 18.7 hours.

No complications outside of the symptoms reported above were noted, and all of the patients made complete and uneventful recoveries.

LABORATORY DATA

Specimens of watery, foul-smelling stools were submitted for examination. None contained mucus or gross blood. Plating on the usual media for the isolation of enteric pathogens gave no unusual organisms of the Salmonella, Shigella or Proteus groups.

A composite mixture of the foods served on the cold plate of July 6 was obtained. These were separated out as far as was possible and cultures made from portions fairly free of other ingredients. Jellied chicken, the peas in the jellied chicken, the salad dressing and egg yolk were cultured on eosin methylene blue plates and on blood agar. Smears made from these specimens showed a few sporulating gram positive bacilli, rare gram negative bacilli, and numerous gram positive cocci, singly, in pairs and in small groups. No pathogens of the enteric series were isolated from the plates. On blood agar, almost pure cultures of *Staphylococcus aureus* were obtained from each of the ingredients. The growth was profuse, between 150 and 300 colonies being present on each plate. Of these the following percentages were *Staphylococcus aureus*:

Salad dressing	90.3%
Jellied chicken	72.6%
Peas from jellied chicken	73.0%
Egg yolk	86.0%

All these strains, as well as those subsequently recovered from the nose and throat of the carrier, probably responsible for the contamination of the food, were hemolytic on blood agar, formed golden yellow pigment, both on blood agar, and on bromthymol blue agar, fermented mannitol, and produced coagulase; all of which are indicative of the pathogenicity of the organism.

One of us examined the three persons engaged in the preparation of this food. None of the three showed any skin lesions which might have been the source of the staphylococci. Nose and throat cultures were taken on July 8. Mrs. X, who had picked over the entire lot of chicken and assisted in the preparation of both the chicken salad and jellied chicken, had a pure culture of *Staphylococcus aureus* in her nose and a large number of the same organisms in her throat. Mrs. A, the caterer, had three colonies of this organism in cultures from her throat but none in her nose cultures. The third member of the personnel failed to show the presence of the organism.

The carrier, Mrs. X, had had sinus trouble for a number of years and suffered from eighth nerve deafness. Positive cultures were obtained on July 8 and 12. Following this an intensive regime of treatment under the care of a rhinologist resulted in one negative culture on July 20. Despite the treatment, however, the cultures taken on August 12 and September 22 were again positive.

DISCUSSION

The evidence outlined above allows us to summarize the probable chain of events. Mrs. X, while showing no symptoms, was a nasal and faucial carrier of virulent *Staphylococcus aureus*. In preparing the chicken on July 5, she contaminated this ingredient as well as the salad dressing. The chicken salad served five to six hours after its manufacture failed to produce symptoms in the guests at the wedding reception because the organism had not had time to produce a sufficient quantity of its enterotoxin. One portion, put aside at between 45°F. and 50°F., and eaten 54 hours after its preparation, produced typical illness in the one individual consuming it. Refrigerator temperatures, unless below 40°F., do not inhibit the growth of this organism or the elaboration of its toxin.

Either the jellied chicken or the salad dressing, or both, which were served on July 6 were similarly contaminated. Both these ingredients were

kept in an ice-box for 24 hours before being served, and at atmospheric temperature for two hours immediately prior to their use. The remaining items on the menu of July 6 may be dismissed with a brief comment. The butter used was made from pasteurized milk. The cottage cheese was likewise prepared. Both these materials were picked up from the dairy about an hour before the luncheon. No other cases of food poisoning were unearthed by a canvass of the local hospitals and physicians, and the samples of these two items served at the luncheon represented only a small portion of the lots made up and sold by the dairy. None of the other items on the menu could be considered seriously.

Rough estimates of the number of *Staphylococcus aureus* in the salad dressing, based on the count per oil immersion field, placed these organisms at from 5,000,000 to 8,000,000 per cc. It should be remembered, however, that the sample was picked up 24 hours after the luncheon of July 6, and that the organisms had had an opportunity to increase considerably during that period. Likewise, little significance can be attached to the relative proportion of the organisms in the various ingredients of the menu, not only because of this extra period of growth, but also because of the impossibility of entirely separating the ingredients from the composite sample submitted.

The symptoms of those attacked were entirely compatible with the diagnosis of staphylococcus food poisoning. The relatively short incubation period, the sudden onset, the short though severe nature of the illness, and the rapid and complete recovery are characteristic of this type of epidemic. The variations seen in individual case histories are those usually encountered and are undoubtedly due to the varying amounts of the infected food consumed and to the imponderable factor of individual susceptibility.

We would lay particular stress, as we did in our opening paragraph, on the prompt reporting of these cases which enabled us to secure specimens of the foods served before they were discarded. All too frequently delayed reporting prevents the examination of these samples which furnish the connecting link between carrier and outbreak, and which also give much needed valuable information, not only on the types of food which may be suspected in future outbreaks, but also on the defects in food handling which made such outbreaks possible.

SUMMARY AND CONCLUSIONS

1. An epidemic of food poisoning due to *Staphylococcus aureus* is described. A carrier, harboring these organisms in her nose and throat, probably infected either home-made boiled salad dressing or chicken, or both.

2. The consumption of this infected food within five or six hours of its preparation caused no symptoms in a group of 45 individuals.

3. One portion of this food, consumed after 54 hours at refrigerator temperature, produced typical symptoms in the one individual eating it.

4. Both the chicken and the salad dressing prepared at this time were served 26 hours later to a group of 70 adult ladies. Of 61 who were interviewed, 43 showed, in varying degree, symptoms of food poisoning.

5. Laboratory examinations of the food served at this luncheon were made possible only by the prompt reporting of the cases.

6. Despite intensive treatment by a specialist, the carrier of *Staphylococcus aureus* believed to be responsible for this epidemic still continues to show *Staphylococcus aureus* in nose and throat cultures.

LONGEVITY OF FUNGI ON CARRIERS

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The evaluation of germicides (fungicidal, bactericidal, virucidal agents) is a problem of special attraction. It involves a host of theoretical and practical questions. Innumerable experiments have been performed, standard methods have been established, and yet new questions and problems arise almost unceasingly. The expression "new questions" does not refer primarily to the testing of recently invented or discovered germicidal substances, though in this field too fundamental findings are liable to occur. In this connection it may suffice to mention the chemical disinfectant zephiran chloride[2] and the antibiotics produced by micro-organisms.[3]

"New questions" refers rather to the fact that the solution of one problem in the field of disinfection very often gives birth to one or more new problems. It has been said that, if the solution of a problem engenders a new question, the first problem was a sound one. It seems then, that the development in the field of disinfection is on the right way.

Sometime ago it became necessary to devise a method for testing fungicides, since the then known methods were not satisfactory. The procedure has been described in this journal.[1] The technic is to infect suitable carriers (cotton strings, filter paper, etc.) with the fungus to be tested. The special way of infecting the carrier insures that the fungus adheres to the carrier without losing its natural contexture.

As mentioned in the former communication,[1] it was planned to test the longevity of fungi adherent to carriers. These experiments have now been performed with the following fungi: Trichophyton gypseum, Microsporon audouini, Microsporon lanosum. The results are given in the tables below.

The results with Trichophyton gypseum and Microsporon lanosum demonstrate that these fungi adherent to carriers remain alive for at least several months; Microsporon audouini yielded somewhat inconsistent results. However, the period of survival extended to 276 days.

It seems that a certain amount of moisture enhances the viability of the fungi on the carrier.

In the course of our disinfection experiments with fungi, we have observed peculiar changes in the growth of Trichophyton gypseum. The cause of these phenomena is under investigation, and the results will be reported in due time.

TABLE 1
Trichophyton Gypseum

Carrier	Days after infection, when tested	Result
Cotton string	102 days	Growth
" "	119 "	"
" "	239 "	"
" "	253 "	"
Filter paper	283 "	"
Linen	314 "	"
Cotton string	346 "	"

TABLE 2
Microsporon Audouini

Carrier	Days after infection, when tested	Result
Cotton string	78 days	Contaminated
" "	180 "	Growth
" "	217 "	No growth
" "	276 "	Growth

TABLE 3
Microsporon Lanosum

Carrier	Days after infection, when tested	Result
Cotton string	102 days	Growth
" "	138 "	"
" "	180 "	"
" "	235 "	"

SUMMARY

Experiments for testing the viability of fungi adherent to carriers have revealed that Trichophyton gypseum, Microsporon audouini and Microsporon lanosum may survive for at least several months. This fact offers the possibility of shipping infected

carriers to various places. This involves two advantages: (1) easier method of shipment, and (2) the possibility of performing similar experiments at distant places with identical fungus material.

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A Message from the Surgeons General of the Army and Navy to the Physicians of the United States

On March 18, 1944, the Emergency Maternity and Infant Care program for the wives and infants of enlisted men in the four lowest pay grades of the armed forces of the United States will have completed its first year. Approximately a quarter of a million wives and infants will have been given care under the program. More than 90 per cent of this number are wives of enlisted men; nearly 10 per cent. are their newborn infants. Medical, nursing and hospital care is being made available in army and navy installations where it does not interfere with the care of the soldier and where it can be given without increasing existing facilities. Whatever other care is available in the place where the wife and infant are living is being given through the civilian authorities.

Physicians the country over are contributing their medical skill to this wartime program generously and in return for moderate recompense. Hospitals the country over have opened their doors to these wives and their infants making available accommodations where their medical needs can be met adequately, though without luxury care. Nurses the country over are helping in the city and the rural homes and in the hospitals.

All this is being carried out voluntarily by those who are participating in the program. All this is being done in spite of the great shortage of physicians and nurses serving the civilian population—a shortage caused by the entry into the armed forces of thousands of our physicians and nurses.

This program of maternity and infant care for wives and infants of enlisted men is made possible by grants from the Federal Government through the Children's Bureau of the Department of Labor and the State health agencies, for the purpose of relieving anxiety among the enlisted men as to how the costs of maternity care for their wives, or the costs of medical care for their infants, will be met in their absence from home while in the armed forces—when, for a great majority, their family income has been lowered materially. The program carried out by the State health agencies brings assurance to the enlisted men that their national and State governments are doing whatever is in their power to make care available to their wives and infants, that physicians throughout the country are helping.

The morale in the armed forces is being raised and our fighting men go overseas with greater confidence in the security of their families because of this wartime program.

We who are responsible for the health and medical care of the men in the armed forces are grateful to you—physicians, nurses, and hospitals—who are participating in this program of care for the wives and infants of these men. You are sharing with us our normal peacetime responsibility of caring for the families of our men, and so are making it possible for us to give our best efforts to the men themselves.

Your contribution is an invaluable aid to us in the prosecution of the war, and we count on your carrying this program forward in the year to come with the same generous spirit you have shown in the past year.

(signed) ROSS T. MCINTIRE,
Vice Admiral, M.C., U.S.N.,
The Surgeon General of the Navy.

(signed) NORMAN T. KIRK,
Major General, U.S.Army,
The Surgeon General.

THE ARMY SPECIALIZED TRAINING UNIT AT THE MEDICAL COLLEGE OF VIRGINIA

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GENERAL

There are now approximately 300 medical and dental students in the Army Specialized Training Unit at the Medical College of Virginia. For a number of years there has been an ROTC Unit in the School of Medicine, which still exists on a much reduced scale. On June 3, 1943, the 3313th Service Unit (ASTU) was activated at this school, and physically qualified medical and dental students, including most of those in the ROTC Unit, were inducted into the Army, processed at Camp Lee, and transferred back to the College as members of the ASTU. By contract with the Government, the Schools of Medicine and Dentistry agreed to furnish professional instruction to certain enlisted men in the Army. However, in this case, these enlisted men happened to be men who were already medical and dental students in the College, who were immediately inducted into the Army, and who are to continue in school as enlisted men until graduation, or as long as the contract is in force.

SELECTION OF TRAINEES*

As stated above, the original members of the Unit were students already in college. Those members of the Unit who enter the classes starting December 30, 1943, and October 4, 1944, are men who are selected by the College, just as the College has always selected its students.

Civilians selected by the college for Medicine or Dentistry who possess evidence that their orders for pre-induction physical examinations were dated prior to March 1, 1944, can expect to be transferred to this college in October, 1944.

Soldiers on active duty who have letters of acceptance dated prior to April 1, 1944, from schools of Medicine or Dentistry can expect to be transferred to the college for the October, 1944, class, provided they have not been alerted for overseas shipment. Provision is also made for students taking pre-medicine or pre-dentistry while on an inactive status as enlisted reservists or who hold commissions in the Medical Administrative Corps to be transferred to A.S.T.P. when accepted by the college for the October 44 Class.

During the period between entry into the service and transfer to the college in October, 1944, eligible students are attached to Army Medical Installations in the vicinity of the college. Most of these men are in hospitals or dental clinics receiving Army Medical Department Training.

Starting with the first class in 1945, students for professional training in the ASTP will be selected by Army Selection Boards from among those students now in pre-medical A.S.T. Units. These Selection Boards will consist of Army personnel and civilian representatives from the various Medical Colleges, who will act as consultants, and who have the power of veto over any applicant. Since there are a great many more students taking pre-professional training in Army Specialized Training Units than the number of vacancies existing in Medical and Dental Colleges, there will be a great deal of competition for the appointments. Starting with the 1945 class, the quota planned in the medical classes is an allotment of 55 per cent Army, 25 per cent Navy, and 20 per cent civilian students. Civilians must be deferred under current selective service regulations.

MILITARY STATUS

Trainees in the Unit have the same military status as any soldier, and they are governed by Army Regulations, the Articles of War, and are subject to Courts-Martial. They are detailed to the Unit as a duty assignment, under the direct command of the Commandant of the Unit. Each is a Private First Class, whose term of service, under present laws, is for the duration plus six months. Trainees are under no more nor less obligation to the Government than any other soldier who is enlisted for the duration plus six months.

The Unit is administered by the Professor of Military Science and Tactics, in his capacity of Unit Commandant. The Commandant is assisted

*Policy as of April 14, 1944.

by one Captain, one First Lieutenant, one Second Lieutenant, and seven enlisted men. These overhead personnel are known as the Permanent Cadre. The Commandant is responsible for fulfilling the best interests of the Government, the College and the Trainees. He must see that the contract between the College and the Government is fulfilled. Under Army Regulations, the assignment of the P.M.S.&T., who is also the Commandant, is a function of the Service Command, and "All details of officers at civil educational institutions for duty with units of the Reserve Officers Training Corps will be made with the concurrence of the institutional authorities concerned."

Discipline, with the trainees, is not a problem. There are very few military requirements other than prompt attendance at classes and the military courtesy prescribed for all soldiers.

SEPARATION FROM THE UNIT

The Contract between the College and the Government provides that "The War Department may withdraw any Army trainee at any time it may elect to do so, and it will do so upon the written request of the Contractor (College) in the case of any Army trainee who fails to meet the academic requirements of the Contractor." In the past, those students who have been separated from the Unit prior to graduation have been sent to a Medical Replacement Training Center, where they are retained in the Medical Department of the Army as enlisted men. Students are not transferred to other duties in the Service before graduation, except for academic failure, or when transfer is obviously justified.

There are two reasons why the percentage of academic failures should be lower among Army trainees than the percentage of failures among civilians in peacetime. First is the freedom from financial struggles, and the second is selection of men who meet Army physical requirements.

At the time of graduation, trainees are discharged from the Service as enlisted men, and commissioned as First Lieutenants in the Medical or Dental Corps. The Dental graduates are called to active duty immediately, provided vacancies exist in the Dental Corps. All of the Dental Class graduating December 18, 1943, received orders to a Medical Replacement Training Center, effective three or four weeks later, allowing time for the State Board examinations.

Medical Graduates are commissioned, and placed on an inactive status until the completion of their internship, which is now a nine month internship, preferably rotating. At the end of the nine month internship, some are given additional deferment for residencies, and the remainder are called to active duty.

DAILY ROUTINE

There are no Army Barracks at this Medical college, and trainees are paid a monetary allowance in lieu of rations and quarters. They may select a place to live anywhere within reasonable commuting distance of the College. Most trainees live in private homes or fraternity houses, as they have always done.

The Unit is organized on the Cadet system. By that System, selected trainees are given Cadet rank from Corporal to Captain, and as such are entitled to the respect and exercise of authority commensurate with their rank. Cadet officers are frequently those trainees who have had previous military training, and many are graduates of Virginia Polytechnic Institute, Virginia Military Institute or the Citadel, and one student in the December 1943 Class was a graduate of the U. S. Military Academy at West Point, with two years of service in the Regular Army as a commissioned officer.

The class rolls are checked in each class for those absent or late. The Senior Cadet Officer in each class is responsible for this duty. Prompt attendance at all classes is strictly enforced. Students realize that spending thirty-six months as a medical student at Government expense is quite different from spending that time in a fox hole, and most of them are putting forth their best effort.

The proper rendering of all military courtesy is emphasized. That gives the men valuable training, and pride in their military status, without requiring time from their studies.

Each student receives one hour of class room instruction per week in Military Science and Tactics, and in the functions of the Medical Department. This instruction is very similar to that given the ROTC students in the past, with emphasis on the military changes of modern warfare. In addition to the military class room work, the Unit has an average of one hour per week of military formations, such as close order drill, road marches, or a trip to

the Richmond Air Base to go through the gas chamber, etc. These outdoor periods are customarily held on Saturday afternoon, and are for the dual purpose of training and mental relaxation. Physical training for medical units is optional with the local Commandant. Physical training is needed by medical students, but no suitable time or place has been found in the crowded schedule of this school.

All military activities are necessarily limited by the demands made on the time of the students by academic requirements.

BENEFITS TO TRAINEES

Benefits to trainees consist of all those benefits received by any soldier, plus the opportunity to complete a four year professional course during time of war. Nothing has been added to or subtracted from the professional curriculum, except the summer vacation, which a few colleges had subtracted prior to the wartime accelerated program.

The financial advantages to the students are considerable. Trainees are furnished tuition, uniforms, textbooks, instruments, and medical care in the Medical College Hospital at Government expense. The actual pay and allowances of an unmarried trainee is $138.55 for a 31 day month. For a married trainee with one child, the pay and allowances for a 31 day month is $196.55, with $20.00 more per month for each additional child. A married trainee with no children receives $166.55 for a 31 day month. Of course, they are also entitled to Government insurance, and, after discharge from the Service, to any pension or medical care for veterans due any soldier. Shortly after activation of the Unit, there was quite an epidemic of weddings. Medical and Dental students had suddenly become quite desirable, which is a radical change from the "old days", as all alumni can verify.

We are proud to say that members of the Unit contribute very generously to the Red Cross, Community Fund, and War Bond Campaigns. During the month of September 1943, members of the Unit made cash purchases of War Bonds totaling $7,341.25. This was in addition to those purchased monthly by allotment from their pay.

The one advantage the students appreciate most about the Unit is the privilege of being in uniform. After a young, healthy man has walked the streets of Richmond in civilian clothes for a year, while his country is at war, and has constantly faced the public and his friends with an apology, he appreciates the honor of being in uniform. He opens his books at night with the satisfaction of knowing that he is serving his country where he is needed most.

Fine Dispensary Serves United States Troops Abroad.

So that United States Army personnel can have the best possible medical attention, two general dispensaries in the London area were recently combined to form the most complete overseas dispensary in the world.

This medical installation was the first one set up in the British Isles. In 1942 the unit was both a hospital and dispensary, treating medical, surgical, and lying-in cases alike, but as Army General Hospitals were installed, this dispensary eliminated its wards until now it specializes in medical, light surgical, therapeutic treatment, and laboratory and x-ray diagnosis. Every doctor at this dispensary is a specialist in his field.

The dispensary gives regular medical attention to officers, enlisted men, WACs, civilians working for the Army, and members of the casts of USO Camp Shows. Besides, in emergencies, civilians and others who are air raid casualties are tended. There is a woman doctor and four WAC assistants who attend WACs and civilian women working for the United States Army.

Classes are conducted by specialists on the staff for other officers and enlisted men attached to the dispensary to further add to their medical knowledge and so help keep the American soldier the "best taken care of in the world".

PUBLIC HEALTH

I. C. RIGGIN, M.D.,
State Health Commissioner of Virginia

The report of the Bureau of Communicable Diseases of the State Department of Health for March, 1944, as compared with the same month in 1943, and for the period of January through March, 1944, compared with the same period in 1943, follows:

	Mar. 1944	Mar. 1943	Jan.-Mar. 1944	Jan.-Mar. 1943
Typhoid and Paratyphoid Fever	8	8	17	36
Diarrhea and Dysentery	139	142	474	321
Measles	4,630	2,451	8,638	4,060
Scarlet Fever	413	202	921	611
Diphtheria	12	30	62	123
Poliomyelitis	1	2	2	10
Meningitis	98	145	254	385
Undulant Fever	4	0	10	6
Rocky Mountain Spotted Fever	0	0	1	1
Tularemia	8	4	16	23

DIARRHEA, DYSENTERY AND ALLIED SALMONELLA INFECTIONS IN VIRGINIA

It is not the purpose of this statement to discuss technically the complexities encountered in the routine examination of fecal specimens submitted to the State Health Department. The object is to acquaint the medical profession with what is being accomplished especially with the Salmonella group of organisms.

In 1898 Shiga established by bacteriological methods the cause of bacillary dysentery which bears his name. By self-inoculation of killed cultures of his organism, he proved that the organism was responsible for the occurrence of a severe type of dysentery. This form oftentimes is found in Japan and is accompanied by a high mortality rate. As is known, this type of bacillary dysentery has since been found in many other parts of the world.

Two years later Simon Flexner, in the course of an investigation of tropical diseases in the Philippines among American troops, discovered an organism associated with dysentery which he believed to be similar to the one described by Shiga. Further work on this organism by Strong and Musgrave indicated that Flexner's organism was not identical to the one found by Shiga, but to be one of much less virulence and a member of an heterogeneous group capable of causing dysentery and having different biological and serological properties, though similar in many other respects.

Following the classical achievements of the above two investigators, other specific organisms have been found in recent years to be the cause of bacillary dysentery, viz., the Schmitz, Sonne, and New Castle types. Generally speaking, the Shiga type of bacillary dysentery is regarded as being the most virulent of all, probably due to its ability to excrete a soluble toxin which has been found lacking in the other recognized types. Irrespective of the toxin producing ability of the Shiga variety and the absence of toxins in the other types, these organisms sometimes show great variation in the severity of symptoms among similar clinical cases.

In the diagnosis of dysentery and gastro-enteritis, the importance of the toxins in botulism and staphylococcus infections must be borne in mind. In staphylococcus contamination of food there is no satisfactory laboratory procedure whereby one can determine when the usually unimportant strain of this variety may flare up and develop a highly virulent toxin.

To the above established causes of bacillary dysentery may now be mentioned a group of organisms of doubtful etiology. These organisms frequently are found in the stools of persons suffering from diarrhea with many of the typical symptoms of bacillary dysentery in which none of the specific incitants are discovered in the stools. They are known as the Proteus Morgani, Alcaligenes, Alkalescens and Pyocyaneus bacilli.

In addition to the bacillary agents of infection, may be added two recognized species of Protozoa, Endameba Histolytica and Giardia Lamblia.

All of the aforementioned known and suspected causes of dysentery have been found in Virginia. It is of interest to note that the Shiga type of bacillary dysentery appears to remain localized in the southwestern section of the State. No isolation of this type in the eastern portion of the State is known regardless of native migration from the Southwest to other areas.

There remains the Salmonella, or so-called "food poisoning group" of organisms to be considered in

enteric fevers and gastro-enteritis infections. Heretofore this group, with the exception of typhoid and the paratyphoids, has been relatively obscure. The infections by this group have not shown any particular decrease or increase. Recognition is made possible due to the perfection of selective laboratory media and refinement in serological methods of specie identification. Most credit is due to the recent brilliant attainment of Kauffmann and White in developing specific agglutinating serum for each variety of organism in this group. The fourth revision of Bergey's classification of the Salmonella group of organisms published in 1934 describes twenty-one species. Cultural requirements listed in this volume and used as an aid in the identification of members of this group are now known to be of little value. The serological methods in use at that time for recognition of the individual species were difficult and tedious procedures because certain agglutinins had to be removed from some types of sera prepared with one variety of the group to prevent cross agglutination with other members of the group, all of which are intimately related in antigenic structure.

With the introduction of the Kauffmann-White procedure the confused status of the group has been reduced to that of a well defined and less erring method of identification. Confirmation lies in the fact that since 1934 the present known types found in various parts of the world have increased from twenty-one to more than one hundred.

Much credit for subsequent work in identifying and classification of strains from various sections is due Edwards and Bruner* of the University of Kentucky. These men conduct a Salmonella typing center and have recently published their results of a study of 3,090 cultures of this group. These strains were found to represent 59 specific types and were obtained from the following sources, fowls 1,828, horses 53, ruminants 20, swine 476, carnivores 90, rodents 90, and from man 532.

The examination of the 532 Salmonella cultures of human origin by Edwards and Bruner covers a seven year study of cultures submitted to them from various parts of the United States and its territories; it represents 41 of the 59 total Salmonella species found by them in all varieties of fowls and animals. Of the human fecal isolations, 123 cultures were derived from normal carriers, 225 from cases of gastro-enteritis, and 86 from enteric fevers. Forty of the 532 cultures were obtained by blood culture. The authors note that the figures do not give a true picture of the relative incidence of infection by the various types of the Salmonella group, since some of the more easily identified varieties are recognized in most public health laboratories and are therefore not represented in their study. A brief discussion of the prevalent types encountered follows:

The common para "B" typhoid organism is represented by one culture from a case of gastro-enteritis, and by 58 cultures from enteric fevers; while the closely related Java variety of the same organism constitutes 30 cultures from gastro-enteritis cases, and none from cases of enteric fevers. S. typhi-murium, commonly known as B. aertrycke and found to be most prevalent in fowls, is represented by 43 cultures from cases of gastro-enteritis, while the Copenhagen variety of the same organism is found in only two similar cases. The authors believe that the majority of human infections caused by the Copenhagen variety can be traced either directly or indirectly to infected pigeons.

S. cholera-suis (so-called American type of B. suipestifer) though not represented in their study from fecal isolations, was present in two blood cultures in contrast to their findings of six cultures of the Kunzendorf variety (commonly known as the western European type) from fecal specimens of gastro-enteritis and also from 17 blood cultures, four of which were fatal cases. Eighteen cultures of S. Newport were found in carriers as against 31 isolations from fecal material in cases of gastro-enteritis. S. Newington occurred 31 times from carriers and in five cases of gastro-enteritis. From the total cultures (369) submitted from persons suffering from all types of infection, 20 known deaths had occurred.

A miscellaneous group of 15 cultures from human sources is described as follows: gall-bladder infections 6, blood stream isolations in endocarditis 2, spinal fluid in meningitis 3, hepatic abscess and shoulder-joint infection 1 each, peri-renal abscess and urine isolation in prostatic involvement comprise 1 each.

S. anatum regarded as the causative organism in the disease "Keel" of ducklings was found in seven cases of gastro-enteritis, while S. pullorum associated with "white diarrhea" of chicks was represented

by one culture.

The identification procedure requires the immunization of sixty or more rabbits in the preparation of agglutinating sera. From these many others are prepared by the absorption of certain antigenic components from portions of the original serum.

During the past eighteen months, the bacteriological feces division of the Virginia State Health Department Laboratory has reported eighteen strains of this group, one of which was from a carrier. Nine specific varieties are represented by these strains which were typed by Dr. Edwards. Preliminary procedures seem to indicate that Salmonella infection in man in Virginia may be more prevalent than heretofore believed.

*J. Infect. Dis. 72:58, 1943.

CASE REPORT OF MATERNAL DEATH

MATERNAL HEALTH COMMITTEE
MEDICAL SOCIETY OF VIRGINIA

The patient was a twenty-five year old colored multipara who had had two previous pregnancies. There was no prenatal care. At the ninth month of pregnancy bleeding began and the following day a midwife saw the patient, who was still bleeding. A physician was called who found the patient in active labor. A spontaneous delivery of a stillborn child occurred shortly after his arrival. Bleeding had continued throughout the labor and after delivery. The placenta was retained. Pituitrin was given but from the information available to the committee it is not clear when, if ever, the placenta was delivered. A consulting physician saw the patient also, and suggested hospitalization. The bleeding continued, and the patient died about three hours later before the arrival of an ambulance.

COMMENT

This case has been classified by the Maternal Health Committee as a preventable obstetrical death, because of the absence of prenatal care, and neglect on the part of the patient and her family in obtaining medical attention at the onset of the bleeding. In addition, it is felt that the patient after the arrival of the physician should have been referred to a hospital where an attempt could have been made to replace the blood loss by transfusions. Of course, it is not known whether the patient had a placenta previa or a premature separation of a normally implanted placenta, but actually it makes but little difference which was present—as long as the blood loss was replaced. The golden hours for treatment in this patient was the first day of the bleeding even before the arrival of the midwife. For this the patient and her family were at fault. Hospitalization at that time would have given an opportunity for diagnosis and preparations for delivery and transfusions could have been made. Even on the day of delivery hospitalization and adequate treatment for antepartum, intrapartum, and postpartum hemorrhage may have saved this patient's life. Because of these factors, it is felt that this must be considered as a preventable death.

COMMUNICATIONS FROM OUR MEMBERS IN SERVICE

From Major Herman W. Farber, MC., 227th Station Hospital, APO 928, c/o Postmaster, San Francisco, Calif.

I would like for you to change the mailing address of my Virginia Medical Monthly to the address listed below.

I am now in ————. The weather here is hot. And the insects are gigantic and vicious. However considering everything we are getting along quite well.

I find that the standard of medicine practiced over here is comparable to the high standards of that practiced at home. The hospitals are well equipped. What we miss over here more than anything else is beer and Coca-Cola. We also miss getting early news from home. Most of our News Broadcasts come from Australia. We can also hear a Jap propaganda station that plays the latest excellent American Swing music.

Tropical diseases at first were a problem to us in the way of diagnosis. However, we are becoming very rapidly accustomed to seeing them and we are finding it easier to diagnose and treat them. Malaria is quite universal over here. The mosquito and malarial control projects have worked wonders and to see a mosquito over here is unusual. We have to sleep under mosquito netting every night, because it doesn't take but one bite of one Anopheles to give you the chills.

With kindest regards to my many friends in the Society and thanking you for your kindness, I am—

* * * * *

From Lt. Douglass D. Fear, MC., 55th General Hospital, APO 137, c/o Postmaster, New York, N. Y.

Will you please change my APO address on your records from 515 or 9648 to 137, c/o Postmaster, New York?

I certainly enjoy receiving the Virginia Medical Monthly.

* * * * *

From Major Kenneth N. Byrne, MC., 222nd Station Hospital, APO 292, c/o Postmaster, San Francisco, Calif.

Please change my address from APO 109, Unit 2, to APO 292 as shown above.

Incidentally the new APO number is the same as my old office telephone number in Lexington before I entered the service three years ago.

* * * * *

From Captain Russell G. McAllister, MC., 304th Airdrome Squadron, Office of the Surgeon, AAB, Alamogordo, N. Mex.

Thank you very much for your kind letter of March 28, which was forwarded me by my wife. It is good to know that the Medical Society of Virginia remembers me, and is interested in my activities, and I am glad to receive the MONTHLY which I devour page by page when it does arrive. How good it will be, after this show is over, to be a human again and practice in Richmond once more!

My present address is above, and we have been here since January, when we moved from Blythe, California. I will certainly advise you of any future change of address, either directly or through my wife.

* * * * *

The above communications were among those received during the past month from some of our members in the Services. Remember, the folks back home are always interested in hearing from you and knowing how you are getting along. How about writing us more often so we may be able to continue this department?

WOMAN'S AUXILIARY
to the
MEDICAL SOCIETY OF VIRGINIA

President—MRS. W. CLYDE WEST, Alexandria.
President-Elect—MRS. PAUL C. PEARSON, Turpin.
Recording Secretary—MRS. C. C. SMITH, Norfolk.
Corresponding Secretary—MRS. N. G. SCHUMAN, Alexandria.
Treasurer—MRS. REUBEN F. SIMMS, Richmond.
Chairman, Press and Publicity—MRS. E. LATANE FLANAGAN, Richmond.

Jane Todd Crawford.

Jane Todd Crawford needs no eulogy on these pages! Her life and heroism are well known to all of us, and speak in glowing terms for themselves.

She blazed the trail for those of lesser stature than herself, and it would seem that if anyone deserves a memorial to perpetuate her name and fame through all the years to come—it is she. Such an undertaking as a memorial of this nature, requires time and thought, and a large expenditure of money.

The Southern Medical Auxiliary is working with these three objectives in mind, and while we await the announcement of their plans with a great deal of interest, can not we all, as loyal Virginians, work with them, and contribute something each year to help the Fund to its ultimate growth?

We are not so much concerned with the time, the place, and the nature of the memorial, as that we have a vital part in its completion. Our State President has asked that every member of the Auxiliary contribute a dime this year to the Memorial Fund.

Please let's make it a hundred per cent and "go over the top" in a way that will be gratifying to Mrs. West, as well as ourselves. At this date, three Auxiliaries have already responded to this appeal.

We know that you are interested in this project. May we count on your cooperation as well?

So far, I have been unable to get any new literature on this subject, and until some comes to hand, may I refer you to the book, "Doctors On Horseback", for some interesting reading on the life and heroism of Jane Todd Crawford.

HELEN P. JACKSON
(MRS. J. WALKER)
Chairman, Jane Todd Crawford Memorial.

Auxiliary Meetings.

NORFOLK

A general meeting of the Woman's Auxiliary to the Norfolk County Medical Society was held March 13, followed by a luncheon at Ames and Brownley's tea room.

Dr. Aurelia Nicholls, who inspects in nine city schools, gave an interesting talk on the duties and problems of the school doctor, and stressed the fact that school doctors do not prescribe for or treat those they examine, but offer suggestions in cases they think need attention.

Since this meeting was the Auxiliary's annual celebration of Doctor's Day, Mrs. R. Bryan Grinnan, chairman of Health Education, presented *Annals of Surgery* to the Norfolk County Medical Society in the name of the Auxiliary, and this was accepted for the Society by Dr. N. F. Rodman, its president.

In the business session preceding the luncheon program, the Nominating Committee was appointed by the president, Mrs. R. M. Reynolds, as follows: Mrs. W. E. Butler, chairman; Mrs. H. W. Rogers, Mrs. James W. Anderson, and Mrs. A. G. Horton. Mrs. C. J. Devine, chairman of the War Fund, reported that the Auxiliary had over-subscribed its quota in the Fourth War Loan by $1,000.00. Mrs. Millard Savage chairman of the Casualty Stations, reported that four of the eleven stations had been closed, and Mrs. Anderson reported the second recreation room completed at Camp Pendleton, and a piano and radio given by members to the Fort Story Hospital. The Auxiliary subscribed $25.00 to the Red Cross.

Mrs. W. Clyde West of Alexandria, president of the State Auxiliary, who was to have been a guest, was prevented from attending by illness in her family.

CLARA P. BROCK,
(MRS. M. F.)
Chairman, Press and Publicity.

NORTHAMPTON-ACCOMAC

The Woman's Auxiliary to the Northampton-Accomac Medical Societies held its regular quarterly meeting at the lovely new home of Mrs. Joseph E. Gladstone at Exmore, on April 4. Luncheon was served to twenty-one members and Reverend W. K. Haddock, pastor of Franktown Johnson's Methodist Churches, who was guest speaker. Mrs. W. Clyde

West, President of the State Auxiliary, who had been invited, was unable to attend.

Mr. Haddock was introduced by the Chairman of the Program Committee, Mrs. W. T. Green, and gave an interesting talk "The Five Tensions in the World Today".

At the business meeting which followed, Mrs. W. Carey Henderson presided. Minutes of the November and January meetings were read, and the treasurer's and committee reports were given. A program to have all practical nurses registered, and a round up of girls for nursing was discussed. Mrs. J. L. DeCormis then gave an interesting account of the Mid-Winter State Board meeting and of Dr. J. M. Hutcheson's talk on bills before the General Assembly.

The next meeting will be an all day picnic at the cabin of Mrs. W. L. Cosby at "Silver Beach" on July 11.

CATHERINE R. TROWER,
(MRS. E. HOLLAND)
Chairman, Press and Publicity.

WARWICK

The Warwick County Medical Auxiliary met at the home of Mrs. W. R. Payne on April 10, at 8 P. M., with Mrs. W. A. Mitchell, the new president, presiding.

The following is a list of newly elected officers for the year:

Mrs. W. A. Mitchell—President.
Mrs. W. R. Payne—Vice President.
Mrs. L. J. Richman—Secretary.
Mrs. Murray Dick—Treasurer.

A luncheon meeting will be held in May.

MARION HOLDERBY,
(MRS. C. E.)
Publicity Chairman.

BOOK ANNOUNCEMENTS

Books received for review are promptly acknowledged in this column. In most cases, reviews will be published shortly after the acknowledgment of receipt. However, we assume no obligation in return for the courtesy of those sending us same.

Essentials of Dermatology. By NORMAN TOBIAS, M.D., Senior Instructor in Dermatology, St. Louis University; Assistant Dermatologist, Firmin Desloge and St. Mary's Hospital; Fellow American Academy of Dermatology and Syphilology; etc. Second Edition. J. B. Lippincott Company, Philadelphia. 1944. xiv-497 pages. Cloth. Price $4.75.

Sulfonamide Therapy in Medical Practice. By FREDERICK C. SMITH, M.D., M.Sc. (Med.), F.A.P.S., Editor of *Philadelphia Medicine*, official organ of the Philadelphia County Medical Society; editor of *The Medical World; etc.* Foreword by GEORGE MORRIS PIERSOL, B.S., M.D., Professor of Medicine, Graduate School of Medicine, University of Pennsylvania; etc. Philadelphia, F. A. Davis Company. 1944. xiii-368 pages. Illustrated with numerous engravings, graphs and tables. Cloth. Price $5.00.

Clinical Lectures on the Gallbladder and Bile Ducts. By SAMUEL WEISS, M.D., F.A.C.P., Clinical Professor of Gastroenterology, N. Y. Polyclinic Medical School and Hospital; Gastroenterologist, Jewish Memorial Hospital, New York. The Year Book Publishers, Inc., Chicago. 1944. 504 pages. Cloth. Price $5.50.

Clinics. Volume II, Number 5, February, 1944. J. B. Lippincott Company, Philadelphia. 266 pages. Single issue price in cloth $3.00.

This issue of *Clinics* is particularly significant as it includes a Symposium on War Medicine—the teaching panels presented at the 51st annual meeting of the Association of Military Surgeons of the United States, held in Philadelphia, October 22-23, 1943. The symposium includes discussions on Chemotherapy, Venereal Disease, Fatigue, Fractures, Plastic and Reconstructive Surgery, War Wounds and Burns, and Gastro-Intestinal Disorders. Original contributions in this issue are: Diagnostic Cardioroentgenography in Army Inductees; A Simple Walking Cast; Burns Incident to War—Measures for their Prevention and Treatment; Management of Purpura; Effect of Shock on Intramuscular Pressures; and Ochronosis: Report of a Case with Alkaptonuria and Melanuria.

Care and Feeding of Children. Revised and Enlarged by L. EMMETT HOLT, JR., M.D., Associate Professor of Pediatrics, Johns Hopkins University; Associate Pediatrician, Johns Hopkins Hospital, Baltimore, Maryland. D. Appleton-Century Company, New York. 1943. xv-321 pages. Cloth. Price $2.00.

Holt's Care and Feeding of Children appears once more revised for the sixteenth time. For any book to go through so many editions justifies its use, for there must have been a great demand for its contents.

This edition has been thoroughly revised and enlarged by Dr. L. Emmett Holt, Jr., the son of the recent author of the book. It is very fortunate that Dr. Holt's son is able to revise this remarkable book in the same capable presentation as his father.

Reading through the book one finds that its contents have been brought up to date with the latest advice on feeding, protection against disease, and the behavior problems which are becoming more numerous due to the modern environment of the child. This book, which on account of its abundance of information for recent parents, has been termed, "the parent's Bible on the care of the infant and child", a term which it rightly deserves.

The revised edition by Dr. Holt, Jr., has kept up the high quality of the book in addition to the information derived from the latest medical scientific knowledge. This book is worthy of every parent's library.

L. E. S.

The Compleat Pediatrician. Practical, Diagnostic, Therapeutic, and Preventive Pediatrics. For the use of Medical Students, Internes, General Practitioners, and Pediatricians. By WILBURT C. DAVISON, M. A., D. Sc., M.D., Professor of Pediatrics, Duke University School of Medicine, and Pediatrician, Duke Hospital; Formerly Acting Head of Department of Pediatrics, The Johns Hopkins University School of Medicine; etc. Fourth Edition. Duke University Press, Durham, N. C. 1943. vi-256 pages and Index. Cloth. Price $4.00.

The fourth edition of the Compleat Pediatrician is welcomed with great satisfaction by those who have found the previous editions irreplaceable.

Daily use of the former edition in recent years has resulted in the inability of finding the most recent facts concerning the sulfonamides and the tropical diseases which are included in this edition. This additional information adds greatly to the book, relieving the reader at times of the disappointment of not finding the information he seeks. This results in his obtaining the fourth edition to replace his somewhat shopworn third edition.

To those physicians who are not acquainted with the advantages of possessing and using the Compleat Pediatrician it might be said that it is a ready handbook presenting all of the essential information that one would need at a moment's notice which renders it very valuable to the practicing physician and will save him some very embarrassing moments for the lack of desired knowledge. The first three editions in sequence have been continually carried in my bag as my handy companion to be followed by the fourth.

L. E. S.

Manual of the Diseases of the Eye. By CHARLES H. MAY, M.D., Consulting Ophthalmologist to Bellevue, Mt. Sinai and French Hospitals, New York; Formerly Chief of Clinic and Instructor in Opthalmology, Medical Department of Columbia University, and Director of the Eye Service at Bellevue Hospital, New York, with the assistance of CHARLES A. PERERA, M.D., Associate in Ophthalmology, College of Physicians and Surgeons, Medical Department of Columbia University, New York; Assistant Attending Ophthalmologist, Presbyterian Hospital, New York. New Eighteenth Edition revised. With 387 illustrations, including 32 plates, and 93 colored figures. Published by William Wood and Company, Baltimore, 1943. Price $4.00.

The new Eighteenth Edition of Manual of the Diseases of the Eye represents the last effort of the author to bring up to date his so-called brain child before his passing in December, in his eighty-third year. It is said that "just two weeks before his death, he insisted on completing the correction of proof of this edition."

The first edition was published in 1900; through the years the book has been translated into a number of foreign languages, the most recent one being the Portuguese. In the production of the last few editions, Dr. May has been assisted by Dr. Charles A. Perera.

Despite the fact that the present edition has not been increased in size nor changed in scope, the material has been carefully revised and in some instances chapters have been rewritten in order to bring the contents up to date, and to include essential new material. The text adheres, however, to its original plan in supplying a general "foundation of ophthalmologic knowledge for the undergraduate student and the general practitioner of medicine." To conform to the latest report of the Committee of the Eye Section of the A.M.A. on Appraisal of Loss of Visual Efficiency, changes have been made in the pages dealing with Compensation for Eye Injuries.

The appendix presents the ocular requirements for admission to the Army, Navy, Marine Corps, Coast Guard and Aviation Services of the United States. This section embraces general induction standards for all services. These standards, however, as set forth, are subject to change depending upon exigencies of the services.

P. W.

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VOL. 71 MAY, 1944 No. 5

The Clinical Significance of the Rh Factor

THE Rh factor, so called because it is found constantly in the rhesus monkey, occurs in 85 per cent of human beings. Persons who are Rh negative develop immune anti-Rh agglutinins when treated with Rh positive blood. A subsequent transfusion with Rh positive blood may lead to a serious or even fatal reaction. This phenomenon is of serious import to an Rh negative woman who marries an Rh positive man. The Rh factor is a dominant Mendelian trait. The Rh positive father may be a heterozygote or a homozygote. If the husband is homozygous, all his children will be Rh positive. If, on the other hand, the husband is heterozygous, the child has a chance of being Rh negative. It seems that the only way to tell if the husband is a homozygote or a heterozygote is to examine the children. If an Rh negative woman has an Rh negative child the father is either Rh negative or a heterozygote.

An Rh negative woman may become sensitized to the Rh agglutinogen by the Rh positive fetus she has in her womb. There is present in the fetal red blood cells an antigen inherited from the father which is lacking in the mother. This antigen in the fetal blood may affect the mother's blood, stimulating the formation of destructive antibodies. It is not clear whether or not an actual mingling of the maternal and fetal blood is necessary for this to take place. These antibodies, like other maternal antibodies, diffuse readily into the fetal circulation where they attack the red blood cells containing the antigens, thereby causing erythroblastosis. Usually it is the Rh antigen that is involved, but not always. Other antigens may occasionally cause erythroblastosis fetalis, which is an immunologic incompatibility between fetus and mother. The important clinical fact is that almost always the Rh factor is the one involved and this factor is not recognized in the usual serological tests. The presence of a child with erythroblasto-

sis is good evidence that the mother belongs to the Rh negative group and also that her blood contains destructive Rh antibodies or agglutinins. Should such a mother need a blood transfusion and should Rh positive blood be used, these antibodies and agglutinins in the mother's circulation would agglutinate and hemolyze the transfused blood, and thereby give rise to severe symptoms such as oliguria, and even anuria.

However, the matter is not as simple as this. There are enough exceptions to make it confusing. Whether the exception proves the rule or not we can not say. An Rh positive mother may develop an atypical anti-Rh agglutinin that may affect the infant. Usually, if an Rh negative mother has an infant that is affected with erythroblastosis, all subsequent Rh positive infants will be affected, but this is not invariably so. Occasionally, such a mother will have a normal Rh positive baby even when she has anti-Rh agglutinins in her serum. A great deal remains to be learned about the whole process. Statistically, the combination of an Rh positive infant and an Rh negative mother occurs in 9.46 per cent of all pregnancies, whereas erythroblastosis fetalis occurs only once in 400 pregnancies.

Boorman, Dodd, and Mollison (*J. Obst. & Gynaec. Brit. Emp.* 51:1, Feb. 1944) have recently studied serologically families with a history of hemolytic disease of the fetus, as they prefer to call erythroblastosis fetalis. They divided the families studied into three groups. Group I contains families in which one or more infants were affected with hemolytic disease. Their clinical and pathological criteria for the diagnosis of hemolytic disease were quite strict. Hydrops of the fetus if born alive had to show anemia and erythroblastaemia, or if stillborn, there must have been evidence of erythroblastosis in the organs. A stillbirth without hydrops, but with widespread extramedullary erythropoiesis was included. Icterus gravis neonatorum occurring within three days of birth, and followed by severe anemia or by death within ten days, and congenital anemia of the newborn occurring within ten days of birth in the absence of any obvious cause, such as sepsis, were counted. Group II contains families with stillbirths and neonatal deaths in which no proved case of hemolytic disease occurred. In group III they put the cases in which the infants had "physiological jaundice".

In group I there were 100 mothers and of these mothers who had had one or more proved cases of hemolytic disease of the newborn, 97 were Rh negative. Ninety-three of these 97 mothers had anti-Rh agglutinins. In the sera of the 3 Rh positive mothers immune agglutinins incompatible with fetal erythrocytes were found in every case. In one case, the mother, father, and infant, were all group A Rh positive, but the mother's serum contained an agglutinin (anti-Rh_2) which acted upon the infant's and father's red cells. The other two Rh positive mothers had immune anti-B agglutinins of high titre.

In 59 of the 70 mothers who had one or more stillbirths or neonatal deaths (Group II) no evidence of iso-immunization to the Rh factor could be found. Eleven mothers of this group had a demonstrable iso-immunization to the Rh factor, which the authors thought was the cause of the infant death.

Six of the 60 mothers who had normal children (Group III) were Rh negative. In 3 of these cases anti-Rh agglutinins were found in the mother's serum. One of these mothers had a previous infant with icterus gravis and the present infant had only 92 per cent hemoglobin and 15 nucleated red cells per 100 leucocytes on the third day. This infant was known to have been perfectly healthy at the third month of life. The infants of the other two mothers showed not the slightest evidence of hemolytic disease.

The main importance of making Rh grouping tests during pregnancy is the detection of Rh negative women so as to insure that if they need a blood transfusion they shall not receive Rh positive blood.

There is no unanimity of opinion as to the kind of blood an infant with hemolytic disease should receive. Boorman, Dodd, and Mollison are of the opinion that Rh negative blood should be used. Reynolds (*Western J. Obst.* 52:103, Mar. 1944), on the other hand, says that whereas the infant's serum and reticulo-endothelial system contain antibodies that continue to act upon the Rh positive red cells, Rh positive blood is indicated, or at least, first Rh negative and then Rh positive blood should be used. However, he quotes Dr. Madeleine Fallon to the effect that it makes little difference which type of blood be used for transfusion.

Longevity

THE worldwide publicity of anti-reticular cytotoxic serum of Alexander A. Bogomolets and his co-workers brings up the subject of natural longevity. Bogomolets himself has investigated the subject in the Soviet Union. His field workers have discovered nearly 30,000 centenarians. In 1938 in Abkhasia, a Soviet with a population about that of Virginia, 80% of which is rural, there were twelve persons between the ages of 107 and 135 years. Bogomolets is fond of referring to a peasant by the name of Shapovski who lived near Sukhum and to whom he introduced Henri Barbusse, the famous French writer, in 1927. "Barbusse was amazed to find the man actually 140 years old, vigorous, active, with a hearty voice and an even heartier appetite. Shapovski's third wife was then 82 and his youngest daughter 26."

From Aristotle to Bogomolets, writers upon longevity have maintained that man should naturally live to about 103 to 105 years, since the natural term of life of an animal is five times the period needed for its development. The two great factors in influencing the length of life are heredity and environment, and of the two, heredity is considered the more powerful. According to Raymond Pearl, environmental circumstances play their part in determining the duration of life, chiefly by influencing the rate at which the inherited endowment is used up. The majority of centenarians have been small eaters. Poverty, within limits is an advantage as it removes the dangers of over-eating, particularly of meat, after the body has reached maturity. The insurance companies are unanimous in the opinion that excessive use of alcohol is harmful. Most of the 1,720 centenarians in James Easton's list have belonged to the peasant class and the skeptically-minded have argued that this is so because there are no records to refute their claims. Few emperors and kings, and only five popes, have attained the age of four score. Physicians occupy a position between the poor and the politically great. Hippocrates is said to have lived to be 109 years old, but few of his disciples have lived so long. It has been aptly said of physicians, "Aliis inserviendo consumunter; aliis medendo moriuntur." William Hotchkiss who died in St. Louis, April 1, 1895, is said to have been 140 years old. His Masonic record was traced back one hundred years and showed conclusively that he was at least 121 years old. Dr. R. Baynes of Rockland, Maine, was practicing medicine at 99. He had an almost perfect set of natural teeth and excellent eyesight. When at home, he never slept in bed but on an iron reclining chair. When William Reynold Salmon of Penllyn Court, Glamorganshire, South Wales, died in his 107th year, he was the oldest known individual of indisputably authenticated age, the oldest physician, the oldest member

of the Royal College of Surgeons, England, and the oldest Freemason in the world. The classical examples of longevity are Henry Jenkins, Thomas Parr and Setrasch Czarten. Henry Jenkins was born in Yorkshire in 1501 and died in 1670, aged 169 years. Registrars of the Chancery and other courts showed that he had appeared in evidence 140 years before his death. Thomas Parr, a poor farmer's servant, was born in 1483. He lived in single blessedness until he was eighty. His first wife lived 32 years, and eight years after her death when he was 120 years old, he married again. When he was 152 years old, the Earl of Arundel and Surray persuaded him to visit the King in London. The journey and the excitement proved too much for him and he died in less than a year. A monument to his memory was erected in Westminster Abbey. Setrasch Czarten was born in Hungary in 1537 and lived for 180 years in one village. He died at the age of 187, or as another authority has it, at 185. In 1895 or thereabouts, there was reported from Vera Cruz in the town of Teluca, where the registers are carefully and efficiently kept, the death of a man 192 years old.

Most of the persons of extreme age have been abstemious in their habits. A few, however, have attributed their age to bad habits. Thomas Wishart of Annandale, Dumfries, who died at the age of 124, chewed tobacco for 117 years. John de la Somet of Virginia was a great smoker and lived to be 130 years old. William Riddell, who died at 116 years carefully avoided water all his life and boasted of his love for brandy.

Of more interest than mere longevity is the preservation of the faculties. Eglebert Hoof, who died at Fishkill, New York, in 1764, at the age of 128, never used spectacles, read assiduously, and retained his memory and senses until his death which was due to an accident. Nicholas Petours, canon of the Cathedral of Constance, died at the age of 137. He was always healthy and vigorous, and celebrated mass five days before he died. Mr. Evans of London was in full possession of his mental faculties when he died at 139 years. R. Glen, a shoemaker of Tacony near Philadelphia, died in his 114th year. He walked to Philadelphia to church every week. There have been several instances of remarkable generative ability in old age. Baron Baravicino de Capelis, the oldest man in Tyrol, died at 104 years. He married his fourth wife at 84. By her he had seven children and she was pregnant with the eighth child at the time of his death. John Gilley who died in Augusta, Maine, in 1813, was born in Ireland in 1690. He came to this country at the age of 60 and married when he was 75 a girl of 18 by whom he had eight children. His wife stated that he was virile until his 120th year.

The most comforting thing about these figures is that many men have displayed worthwhile activity when past four score years. Brougham at 82 and Lyndhurst at 88 could pour forth words of eloquence and sagacity for hours at a time. Landor wrote "Imaginary Conversations" at 85, and Isaak Walton was active with his pen at 90. Chevreul, the chemist, DeLesseps, the canal builder, Gladstone, Bismarck, and Pope Pius IX carried their full mental vigor into old age. Michael Angelo was still painting his great canvases at 89, and Titian at 90 worked with all the vigor of his earlier years. The Doge Dandolo with the aid of the French besieged and captured Constantinople at the age of 83. Newton at 83 worked with the same ardor that animated his middle age. Cornaro was as happy at 90 as at 50, and in far better health at 95 than he was at thirty.

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Societies

Virginia State Board of Medical Examiners.

At the meeting of the Board on December 15-17, 1943, the following were granted certificates:

Dr. Abraham Robert Abarbanel, Arlington.
Dr. Dexter David Abeloff, Boston, Mass.
Dr. John Stanard Archer, Jr., Richmond.
Dr. Wilbur James Baggs, Jr., Norfolk.
Dr. Howard Eugene Baldini, Union City, N. Y.
Dr. Richard Albert Bagby, Temple, Texas.
Dr. Bruno Barelare, Jr., Charlottesville.
Dr. Joseph M. Barker, Arlington County.
Dr. Walter Louis Barnes, Richmond.
Dr. Robert Hardy Barnes, Jr., Washington, D. C.
Dr. William Clayton Barr, Virginia Beach.
Dr. Thomas Grasty Bell, Staunton.
Dr. Frank Smoot Beazlie, Jr., University.
Dr. Morton Emmett Berk, Richmond.
Dr. Walter Dick Bigford, Purcellville.
Dr. Joseph Shelton Bower, Lynchburg.
Dr. Paul Edgar Brady, Newark, Ohio.
Dr. Maurice Miller Bray, Charlottesville.
Dr. Albert Compton Broders, Jr., Madison. Wisc.
Dr. Lenora Virginia Brown, Charlottesville.
Dr. Mary Tom Bunting, Washington, D. C.
Dr. Ellsworth Ferrell Cale, Charleston, W. Va.
Dr. Wayne Calhoon Campbell, Chester, W. Va.
Dr. Fay Ashton Carmines, Richmond.
Dr. George Benjamin Carter, Charlottesville.
Dr. Philip Alexander Caulfield, Washington, D. C.
Dr. John Letcher Chesnut, Erwin, Tenn.
Dr. Charles Milton Clark, Jr., Long Island, N. Y.
Dr. Raymond R. Comess, Charlottesville.
Dr. William Henry Cox, Richmond.
Dr. A. Reynolds Crane, Norfolk.
Dr. James Jefferson Davis, Richmond.
Dr. Kelley King Davis, Charlottesville.
Dr. Wirt Lee Davis, Jr., Atlanta, Ga.
Dr. Walter Frank Daughtrey, Jr., Richmond.
Dr. Jose Angel DeJesus, University.
Dr. Paul Henry Dietrich, Petersburg.
Dr. George Henderson L. Dillard, Cleveland, Ohio.
Dr. Julian Quayle Early, Mt. Jackson.
Dr. H. E. Eldridge, Dunn, N. C.
Dr. Milton Ende, Petersburg.
Dr. Henry Edwin Ernst, Roanoke.
Dr. Peyton Randolph Evans, University.
Dr. William Winfree Farley, Richmond.
Dr. Donald Terrell Faulkner, Blackstone.
Dr. Robert Henry Fennell, Jr., Norfolk.
Dr. Frank Micheal Ferrante, New York, N. Y.
Dr. Thaddeus George Fibich, Arlington.
Dr. George Washington Fishburn, Philadelphia, Pa.
Dr. Walter Cleveland Fitzgerald, Crewe.
Dr. Irving Edwin Fixel, Richmond.
Dr. Frederick John Flynn, Jr., Jamaica, N. Y.
Dr. Charles Phillip Ford, Jr., Richmond.
Dr. S. Oscar Fry, Jefferson County, W. Va.
Dr. John Burtt Fuller, Richmond.
Dr. James Cofer Gale, Waverly.
Dr. James Seibert Gamble, Richmond.
Dr. Ovidio Joseph Giovanelli, Radford.
Dr. Thomas Vance Goode, Jr., Richmond.
Dr. Maston Lewis Gray, New Orleans, La.
Dr. Isa Costen Grant, Richmond.
Dr. William Nelson Greever, Chilhowie.
Dr. Innes Correll Haines, University.
Dr. Gerald Eugene Hartle, Wardensville, W. Va.
Dr. Harper Keith Hellems, University.
Dr. Harry Heim Henderson, Richmond.
Dr. Elizabeth Harman Hill, Charlottesville.
Dr. Gunter Hirschburg, Newport News.
Dr. Robert Arnold Hoffman, Richmond.
Dr. Sarah Huldah Hoover, Richmond.
Dr. Joseph David Hough, Franklin.
Dr. Gwendolyn Sully Hudson, Richmond.
Dr. Robert Steele Hutcheson, Jr., Lexington.
Dr. King Arey Jamison, Blacksburg.
Dr. George Watson James, III, Richmond.
Dr. William Lipscomb Jamison, Manassas.
Dr. Alexander Charles Johnson, Jacksonville, Fla.
Dr. William Richard Kay, Richmond.
Dr. Walter Thomas Kees, Fredericks Hall.
Dr. Francis Robert Kelley, Jr., Richmond.
Dr. Arthur Klein, Richmond.
Dr. Carl Millard Lang, Charlottesville.
Dr. William Gordon Leary, Jr., Cincinnati, Ohio.
Dr. Quentin James Legg, Richmond.
Dr. Charles Emerson Liddington, Norfolk.
Dr. Otis Everette Linkous, Jr., Richmond.
Dr. Marion Cooper Lindel, Seattle, Wash.
Dr. Garnett William Link, Charlottesville.
Dr. Leonard Irving Malis, Ventnor, N. J.
Dr. Shirley Sue Martin, Huntington, W. Va.
Dr. William Douglas Martin, Radford.
Dr. Jaun deDios Martinez-Galindo, Charlottesville.
Dr. Robert Murray Miskimon, Richmond.
Dr. Virgil Robert May, Jr., Richmond.
Dr. Robert Earnest McAlpine, Portsmouth.
Dr. Fred Carlton McCall, Richmond.
Dr. John Graham McCown, Roanoke.
Dr. James Edward McGee, Richmond.
Dr. Edward Thomas McKee, Jr., Norfolk.
Dr. Robert Edward Lee, McNeely, Dallas, Texas.
Dr. Howard Everett Medinets, Perth Amboy, N. J.
Dr. Henry Clarkson Meredith, Jr., Norfolk.
Dr. Frank Ferdinand Merker, Baltimore, Md.
Dr. Woodrow Wilson Mills, Kenova, W. Va.
Dr. Henry Rankin Miller, Washington, D. C.
Dr. Richard O'Brien Monahan, Roanoke.

Dr. Frederic Pott Moore, II, Richmond.
Dr. John William Henry Morgan, Ewing.
Dr. Warren Lodowick Moorman, Salem.
Dr. Robert Lord Morrison, New Orleans, La.
Dr. Edward James Mortell, Richmond.
Dr. John Langdon Moss, Emory, Ga.
Dr. Marion Bailey Murdock, Grand Rapids, Mich.
Dr. Anne Newhall, Brookline, Mass.
Dr. Cardwell Camden Nuckols, Portsmouth.
Dr. Margaret Bess Obenschain, Washington, D. C.
Dr. Ruth O'Neal, Richmond.
Dr. Edwin Johnson Otis, Norfolk.
Dr. Robert Franklin Owen, Alexandria.
Dr. Harold Vernon Palmer, Erwin, Tenn.
Dr. Loren Francis Parmley, Jr., Richmond.
Dr. William Frederick Pohl, St. Charles.
Dr. James Harrison Powell, Petersburg.
Dr. John Marvin Ratliff, Norfolk.
Dr. Charles Holland Rawls, Richmond.
Dr. William Hervey Remine, Jr., Richmond.
Dr. Ashby Turner Richards, Harrisonburg.
Dr. Bill Ballard Richmond, Beckley, W. Va.
Dr. Rufus Martin Roll, Washington, D. C.
Dr. George Samuel Rowlett, Jr., Richmond.
Dr. John Edward Ryan, Washington, D. C.
Dr. Robert Saul Salisbury, Richmond.
Dr. William Callier Salley, Norfolk.
Dr. Maurice M. Sauturian, Clintwood.
Dr. Maurice R. Schlanger, New York, N. Y.
Dr. Donald Conrad Schweiser, Charlotte, N. C.
Dr. Cyril Iredell Sease, Jr., Richmond.
Dr. Chauncey Shumaker, Poweron, Ohio.
Dr. Irving Leonard Silverstein, Richmond.
Dr. Robert William Sjogren, Blacksburg.
Dr. Herman Ivan Slate, Alexandria.
Dr. Charles Everett Smeltzer, Jackson, Mich.
Dr. Howard Elmer Smith, Roanoke.
Dr. Harold William Snodgrass, Glade Spring.
Dr. John Hammitt Sproles, Pocahontas.
Dr. Jay Edwin Stoeckel, Richmond.
Dr. Henry Tuttle Stull, Arlington.
Dr. Helen Wickham Taylor, Charlottesville.
Dr. William Basil Thompson, Arlington.
Dr. John Robert Troxell, Brooklyn, N. Y.
Dr. Jack Lankford Ulmer, Richmond.
Dr. George Vranian, Richmond.
Dr. Mary Martin Wade, Baltimore, Md.
Dr. Frank Alton Wade, Richmond.
Dr. Charles Miller Walsh, Crewe.
Dr. Charles Miller Walsh, III, Crewe.
Dr. Louis Napoleon Waters, Portsmouth.
Dr. Edgar Newman Weaver, University.
Dr. Lee Elias Whitlock, Jr., Norfolk.
Dr. Carrington Williams, Jr., Richmond.
Dr. Mark B. Williams, Richmond.
Dr. Grace Evelyn Wilder, Hampton.
Dr. Harold Eugene Wolfe, Marion.
Dr. Walter Dickenson Woodward, Richmond.
Dr. Nathaniel Henry Wooding, Halifax.
Dr. Paul Zwick, Marion.

HOMEOPATHS:

Dr. John Henry Hoge, Norfolk.

OSTEOPATHS:

Dr. Joseph Trivella Rogers, Waverly.
Dr. Ralph Morton Stokes, Jr., Portsmouth.

The Northampton County Medical Society

Held its regular quarterly meeting at Cape Charles on April the 13th, at which time was held the election of officers for the coming year. This resulted in naming the following: President, Dr. J. M. Lynch, Cape Charles; vice-president, Dr. S. K. Ames, Cape Charles; secretary, Dr. W. C. Henderson (re-elected), Nassawadox; and Censor, Dr. J. E. Gladstone, Exmore.

Dr. W. J. Sturgis of Nassawadox gave a comprehensive resume of advances to date in the use of Penicillin. He stated that, due to its great value to men in the Service, the Government has aided in expanding facilities of biological manufacturers so that it is hoped supplies may soon be sufficient for the release of some for general civilian use. Dr. Sturgis and Dr. H. L. Denoon recently attended a meeting of the American College of Surgeons in Baltimore, where papers were presented on this subject by both civilian and military physicians.

Patrick-Henry Medical Society.

The regular quarterly dinner meeting of this Society was held in Martinsville on April the 7th, at which time delegates and alternates were elected to the State meeting in Richmond. After a short business session, an interesting discussion was given by Dr. Hugh H. Trout of Roanoke on Peripheral Vascular Diseases and a movie was shown to illustrate the treatment of varicose ulcers.

Dr. R. H. Walker and Dr. T. Henry Dickerson, both of Martinsville, are president and secretary, respectively, of this Society.

Medical Society of Northern Virginia.

Dr. H. B. Mulholland of Charlottesville was guest speaker at the Spring meeting of this Society held in Front Royal on April the 14th, his subject being "Some of the Newer Drugs and Their Usage". Other speakers were Drs. L. M. Allen, P. W. Boyd and Leslie M. Bell of Winchester, and Drs. Elizabeth Sherman and W. E. Lynn of Front Royal.

Officers of the Society are Dr. Charles O. Dear-

mont of White Post, president; Dr. B. B. Dutton of Winchester, vice-president; and Dr. J. E. Harris of Winchester, secretary-treasurer.

The Roanoke Academy of Medicine

Held its April meeting on the 3rd, at Hotel Roanoke, under the direction of Dr. E. G. Gill. Addresses were given at this time by Surgeon General Parran of the U. S. Public Health Service, Washington, on "Medical Care of the Civilian Population", and by Rear Admiral Joseph J. A. McMullen, M.C., U.S.N., Washington, on "Recent Advances in Navy Medicine".

Dr. A. M. Groseclose is president and Dr. D. S. Garner secretary.

Richmond Academy of Medicine.

At the March 28th meeting of the Academy, Drs. James F. Blades and Raymond Berger presented a case report on "Diaphragmatic Hernia". Lt. Col. A. O. Hampton, MC., Chief of the Radiological Section, Walter Reed Hospital, Washington, D. C., was a guest speaker, his subject being "A Radiologist's Viewpoint of Pulmonary Infarction".

At the April 11th meeting, a case report on "Pulmonary Actinomycosis Due to *Actinomyces graminis*" was presented by Dr. John P. Lynch. Dr. Harry Gold, a member of the staff of the Cornell University Medical College, New York City, was guest and spoke on "Management of the Failing Heart".

News

Committee of Arrangements for Richmond Meeting.

The Richmond Academy of Medicine has appointed the following committee for the meeting of the Medical Society of Virginia, October 23-25:

General Chairman—Dr. Harvey B. Haag
Vice-Chairman and Entertainment—Dr. Don S. Daniel
Hotels—Dr. William R. Jordan
Finance—Dr. James Asa Shield
Publicity—Dr. E. L. Copley
Meeting Halls—Dr. C. L. Outland
Golf—Dr. Thomas W. Murrell
Ladies—Mrs. A. G. Shetter

Cards have been sent to members asking for titles and abstracts of papers to be presented at this meeting. Please do not fail to send these to the Society's office by May 15, as the Program Committee plans to meet soon after that time to make up the program. Also if you would like to present a scientific exhibit, send the title and information about it to the chairman, Dr. W. Ambrose McGee, 1601 Monument Avenue, Richmond 20.

Component societies have been asked to elect their delegates and alternates before they recess for the summer. You will want to be included in the list to be published in the MONTHLY early in the fall and if your Society has not named its representatives, see that it is done promptly.

This promises to be an excellent meeting and the Richmond Committee is already working hard on plans. Be sure to put these dates on your calendar and be in Richmond on October 23-25.

News from the University of Virginia, Department of Medicine.

A Charles Scott Venable Annual Lectureship in Traumatic Surgery has been established in the Medical School of the University of Virginia. This lectureship is supported by royalties from the sale of an adjustable splint designed by Dr. Charles Scott Venable, Jr. The splint has been made available without royalties or encumbrances to the Red Cross and civilian defense agencies and is now in general use both in the United States and Canada. It is standard equipment for the Canadian National Railway. It is also obligatory equipment for all ambulances in the state of Texas.

Dr. Venable was graduated from the Medical School of the University of Virginia in June, 1900. He is chief of staff of the Nix Hospital, San Antonio, Texas, and the seventy-ninth President of the

State Medical Association of Texas, and President of the American Association for the Surgery of Trauma. Dr. Venable's father was professor of mathematics at the University of Virginia from 1866-1896.

On April 11, Dr. Henry B. Mulholland spoke at the meeting of the Medical Society of Northern Virginia in Front Royal, Virginia. His subject was "Some of the Newer Drugs and Their Usage".

Major Henzykzi Borowski spoke at the University of Virginia on April 16. His subject was "Experiences of a Polish Doctor in Peace and War".

Medical College of Virginia News.

Dr. Lewis E. Jarrett, director of the hospital division, has resigned effective June 1, to accept the superintendency of Touro Infirmary, New Orleans, Louisiana. Dr. Jarrett is an alumnus of the college both of the school of pharmacy and of the school of medicine. He was made director of the hospital division in 1933, succeeding Dr. J. L. McElroy who went to Paris, France, as superintendent of the American Hospital.

Dr. I. A. Bigger, professor of surgery, spoke to the staff of the Woodrow Wilson General Hospital, Staunton, on April 13, on *Drainage of the Pleura with Particular Relation to Chest Injuries.*

Dr. W. T. Sanger, president, attended the conference on rural health under the auspices of the Farm Foundation in Chicago, April 10-14.

Dr. DuPont Guerry, III, has been appointed assistant in ophthalmology. Dr. Guerry has recently opened offices in Richmond.

On April 20-21 Dr. Bela Schick, chief of the department of pediatrics, Mount Sinai Hospital, New York, addressed two meetings at the college. The first was the faculty and staff meeting on April 20, when he spoke on *Recent Advances in Pediatrics,* and the second at noon on April 21, when he addressed the student body, faculty, and invited guests on *Allergy.*

Dr. Sidney S. Negus, professor of biochemistry, attended the annual meeting of the American Chemical Society in Cleveland.

Married.

Dr. Walter Dickenson Woodward, Richmond, and Mrs. Alice Myrtle Nevins, Staten Island, N. Y., April 15. Dr. Woodward is a graduate in medicine at the University of Virginia in December, 1943. He is at present stationed at the U. S. Marine Hospital, Staten Island, N. Y.

Lt. (jg.) Harold Eugene Wolfe, MC., USNR., Marion, and Miss Pearl Thomas Simms, Courtland, March 18. Lt. Wolfe is a graduate of the Medical College of Virginia, class of December, 1943.

Dr. Lawrence O. Snead,

Richmond, was inaugurated as President of the Richmond Alumni Club of Phi Delta Theta fraternity at its ninety-sixth Founder's Day Banquet held recently.

Dr. R. D. Garcin, Jr.,

After service for sometime with the Pacific Fleet, has been placed on inactive duty because of a slight injury, and has returned to his home in Richmond where he is engaged in the practice of internal medicine, with offices at 3216 East Broad Street.

The Richmond Eye, Ear, Nose and Throat Society

Held its regular meeting at the Commonwealth Club on April the 11th, at which time the following papers were read:

Newer Concepts in the Surgical Treatment of Cataracts by Dr. Luther C. Brawner and Dr. Edgar Childrey, Jr.

Practical Observations in Otitis Media and Mastoiditis, Bebore and Since Sulfonamides by Dr. Basil B. Jones.

Dr. P. N. Pastore is president and Dr. C. A. Folkes secretary.

Occupational Therapists Needed.

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Any one interested may obtain the necessary application forms from the Secretary, Board of U. S. Civil Service Examiners, at any first- or second-class post office, or from the U. S. Civil Service Commission, Washington 25, D. C. The title of this form is 57/4006-ABC.

Dr. H. Lee Harris,

Formerly on the staff of the Clinch Valley Clinic Hospital at Richlands, has taken over the offices and practice of the late Dr. V. M. Cox, in Reynolds Arcade Building at Bristol, Virginia. His specialty is diseases of the eye, ear, nose and throat.

Dr. A. L. Van Name,

Who has practiced for several years at Center Cross, announces the removal of his home and office to Urbanna.

Dr. B. C. Grigsby,

Who has for the past nine years been associated with the Blue Diamond Coal Company at Bonny Blue, announces the removal of his office to the Dominion National Bank Building in Bristol, Va.-Tenn., where he will be engaged in general practice.

Promotions in Service.

Promotions have recently been noted for the following Virginia doctors in Service:

To Captain in the Navy:
Dr. C. J. Devine, Norfolk.

To Commander:
Dr. M. T. Rosser, Hillsville.

To Colonel:
Dr. Emmett V. Richardson, Marion.

To Major:
Dr. Kenneth N. Byrne, Lexington.

To Captain:
Dr. Samuel F. Driver, Troutville.
Dr. Russell G. McAllister, Richmond.
Dr. H. M. Price, Martinsville.
Dr. George Andrew Shetter, Richmond.
Dr. Weir Mitchell Tucker, Richmond.

Reading (Pa.) Eye, Ear, Nose and Throat Society.

At its meeting on February 16, at Valley Forge General Hospital, an excellent program was presented by Lt. Col. James N. Greear, Jr., chief of the EENT Section. The program consisted of plastic work on war injuries, a new method of bronchography, rehabilitation of blind members of the Armed Forces, retinal detachment, and injuries to the occipital lobes.

Col. Greear is known to many of our readers, being an alumnus of the University of Virginia, Department of Medicine, and an associate member of the Medical Society of Virginia.

Dr. Edgar A. Bocock,

An associate member of the Medical Society of Virginia, superintendent of Gallinger Muncipal Hospital, in Washington, D. C., from 1927 to 1943, was recently appointed director of Doctors Hospital in that city and entered upon his new duties in March.

Shipping Address for Your Art Exhibit.

Artist-physicians desiring to exhibit their works at the June A.M.A. meeting should ship their works not later than May 20 to: American Physicians Art Association, Room 1302, 308 W. Washington St., Chicago, Ill. Pack carefully and ship by express collect, including $50 insurance.

Mead Johnson & Company have offered to pay the express charges both ways (including insurance up to $50). Art objects exhibited are automatically eligible for inclusion in the next Parergon, as well as for one of the numerous A.P.A. Ass'n. prizes.

Further information may be obtained from Francis H. Redewill, M.D., Secretary, American Physicians Art Association, Flood Bldg., San Francisco, Calif.

Dr. J. A. M. Thompson,

Who had been connected with the medical staff of Blue Ridge Sanatorium, Charlottesville, since October, 1933, on January 1 became clinician with the Tubersulosis X-ray Mobile Unit of the State Department of Health.

The Collection and Transmission of Specimens for Bacteriological and Serological Examination.

A pamphlet giving directions for *The Collection and Transmission of Specimens for Bacteriological and Serological Examination* has been prepared in tabular form, easily accessible, and very useful for students, interns, and practitioners.

It may be obtained free of charge from Dr. Fritz J. von Gutfeld of the Bacteriological Department of the Medical College of Virginia, Richmond.

The Society of Chest Physicians of Virginia

Will meet at Hotel John Marshall, Richmond, Monday, May the 29th. There will be a business session from 10:00 to 10:30 a.m. The following scientific program will start at 10:30:

10:30-12:30 P. M.—Presentation of x-ray films showing interesting chest conditions.
Directed by Dr. C. L. Harrell, Norfolk.

1:00 P. M.—Luncheon—Speaker: Dr. C. M. Sharp, U. S. Public Health Service.

2:00 P. M.
SCIENTIFIC SESSION.

1. Tbucerculosis Discovered in Virginia through Selective Service Examinations—Dr. E. C. Harper, Richmond.
   Discussion opened by Capt. George W. McCall (MC).
2. The Effect of the Present World War on Tuberculosis—Dr. Frank B. Stafford, Blue Ridge Sanatorium.
   Discussion opened by Dr. Dean B. Cole, Richmond.
3. Pneumonectomy in Pulmonary Tuberculosis—Dr. Everett I. Evans, Richmond.
   Discussion opened by Dr. M. L. White, Jr., University.
4. The Indications for Terminating Pneumothorax—Dr. Elizabeth C. Cole, Norfolk.
   Discussion opened by Dr. L. R. Broome, Catawba Sanatorium.
5. Supervision of Pneumothorax Cases—Dr. R. H. Walker, Martinsville.
   Discussion opened by Dr. T. N. Davis, Lynchburg.

Lunch will be served for $1.50 per plate at Hotel John Marshall.

Dr. Frank B. Stafford of Blue Ridge Sanatorium is president and Dr. E. C. Harper, State Director of Tuberculosis Out-Patient Service, Richmond, is secretary.

Dr. Leon Culbertson,

Class of '36, University of Virginia, Department of Medicine, and later instructor in urology there, is now assistant professor of urology at Bowman Gray School of Medicine, Wake Forest College, Winston-Salem, N. C.

Arlington County Receives Health Award.

Award of the 1943 National Health Honor Roll to 53 city and county units has been announced by the United States Chamber of Commerce. Among these is Arlington County, Virginia. The awards were based on attainments of high standards in excellence in protecting public health in war time. Emphasis was placed on specific measures to control communicable diseases; sanitation, including protection of milk and food supplies; proper safeguards in maternity care, child health, and welfare; effective health education; adequate industrial health programs and other health services.

New Books.

The following are recent acquisitions to the Library of the Medical College of Virginia and are available to our readers, the only cost being return postage:

A.M.A.—Transactions of the section on Laryngology, Otology and Rhinology. 1940, 1941, 1942.
A.M.A.—Transactions of the section on Ophthalmology. 1940, 1941.
Bachmeyer, A. C. ed.—The hospital in modern society. 1943.
Butterweck, J. and Muzzey, G. A.—Handbook for teachers. 1939.
Christopher—Minor surgery. 1944.
Brown, S. A., Davis, Arthur and Lee, Ulysses—The negro caravan.
Cleveland Child Health Association—Manual for the conduct of classes for expectant mothers.
Cushing, Harvey—Bibliography of Ander Vesalius.
Davis, A. and Dollard, J.—Children of bondage. 1940.
Elliott, H. S.—Process of group thinking.
Estabrooks, G. H.—Hypnotism. 1943.
Gesell, Arnold et al.—The first five years of life. 1940.
Fedorova, Nina—The children. 1942.
Fedorova, Nina—The family. 1943.
Fosdick, Harry—On being a real person.
Glasser, Otto, ed.—Handbook of medical physics. 1944.
Groves, Ernest R.—Marriage.
Hamilton, Alice—Exploring the dangerous trades.
The Harvey Lectures, 1942-1943.
Heer, A. L.—Steps to better teaching.
Hollender, A. R.—Office treatment of the nose, throat and ear. 1943.
Hughes, Langston—Not without laughter. 1941.
Keller, A. G.—Manual of the biochemical laboratories of the Graduate Hospital of the University of Pennsylvania. 1944.

Kisch, Bruno—Strophanthin: Clinical and experimental experiences of the past 25 years.
Law, Frederick M.—Nasal accessory sinuses Roentgenologically considered.
McCay, Clive—Nutrition of the dog. 1943.
Mead, Margaret—From the South Seas.
Coming of age in Samoa.
Growing up in New Guinea.
Sex and temperament. 1939.
Meyer, K. H.—Natural and synthetic high polymers. 1942.
N.E.A.—Department of Supervisors—Cooperation: Its principles and practices.
Newsom, Bryan—Communicable disease workbook. 1942.
Ormsby & Montgomery—Diseases of the skin. 1943.
Osler, William—A way of life. 1932.
Rhinehart, Darmon A.—Roentgenographic technique. 1943.
Richmond, Winifred V.—Personality—Its development and hygiene. 1943.
Salter, Andrew—What is hypnosis? 1944.
Scheinfeld, Amram—You and heredity.
Transactions of American Laryngological Association. 1943. (Including index of subjects and authors.)
Travis, L. E. & Baruch, D. W.—Personal problems of everyday life. 1941.
U. S. Department of Commerce. Bureau of the Census.—Vital statistics of the United States. Part I and II, 1941.
Williams, Rebecca Yancey—Carry me back.

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Obituaries

Dr. Warren Taylor Vaughan,

Internationally known Richmond physician, died April 2, following a heart attack. He was fifty-one years of age and graduated from the University of Michigan Medical School in 1916. Dr. Vaughan served during World War I, during which he rose to the rank of Lieutenant-Colonel in charge of Army Hospital Number 41. Shortly after this, he located in Richmond where he specialized in allergy. In 1936, he established the Vaughan-Graham Clinic which has been outstanding in its work in the treatment of allergic diseases. Dr. Vaughan was the author of several books, and was editor and contributor to many medical magazines. He was a member of numerous medical organizations, among them being the American Society of Clinical Pathologists, the American Rheumatism Association, the Society of Investigative Dermatology, and the International Society of Gastroenterology, and an honorary member of the Institute of the Practice of Medicine of Barcelona, Spain, and the Argentinian Society for the Study of Allergy.

He was also a member of the Beta Theta Phi, Rho Sigma, Sigma Xi, and Alpha Omega Alpha fraternities, and in 1940 he was president of the Harvard Club of Virginia. Dr. Vaughan joined the Medical Society of Virginia in 1920 and was a vice-president in 1931-32. His wife and four sons survive him. Two sons are already practicing physicians and the other two are medical students at Harvard Medical College.

Dr. Samuel Broders Moore,

For many years prominent in the professional life of Alexandria, died March the 15th, after a long illness caused by coronary thrombosis with pulmonary edema. He was seventy-two years of age and a graduate in medicine from Georgetown University School of Medicine of Washington, in 1897. Shortly thereafter he joined the Medical Society of Virginia and took an active interest in its work to the time he had to retire because of his health. He was a former vice-president of this Society, a past president of the Alexandria Medical Society, was a fellow in the American College of Surgeons, and identified with other organizations. He was also for sometime surgeon with several railroads and was chief surgeon to the Alexandria Hospital.

Dr. John Adams Drake,

Monroe, died April 14th, at the age of sixty-three. He was a graduate of the Medical College of Virginia in 1903 and had practiced at Monroe for the past twenty years. Dr. Drake was formerly a member of the Medical Society of Virginia.

Dr. Horace L. Goodman,

Ronceverte, W. Va., died February 28, of heart disease. He was a native of Otter River, Campbell County, Va., and was sixty-seven years of age. Dr. Goodman graduated from the Medical College of Virginia in 1901.

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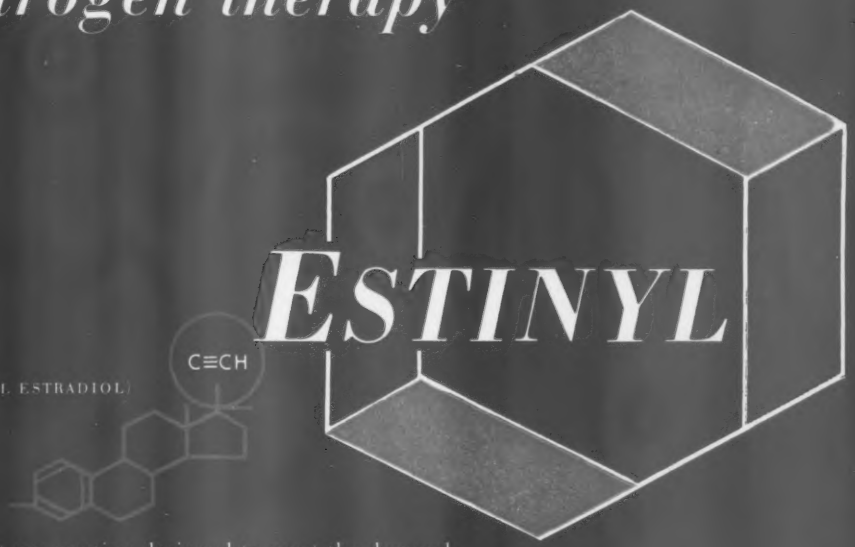

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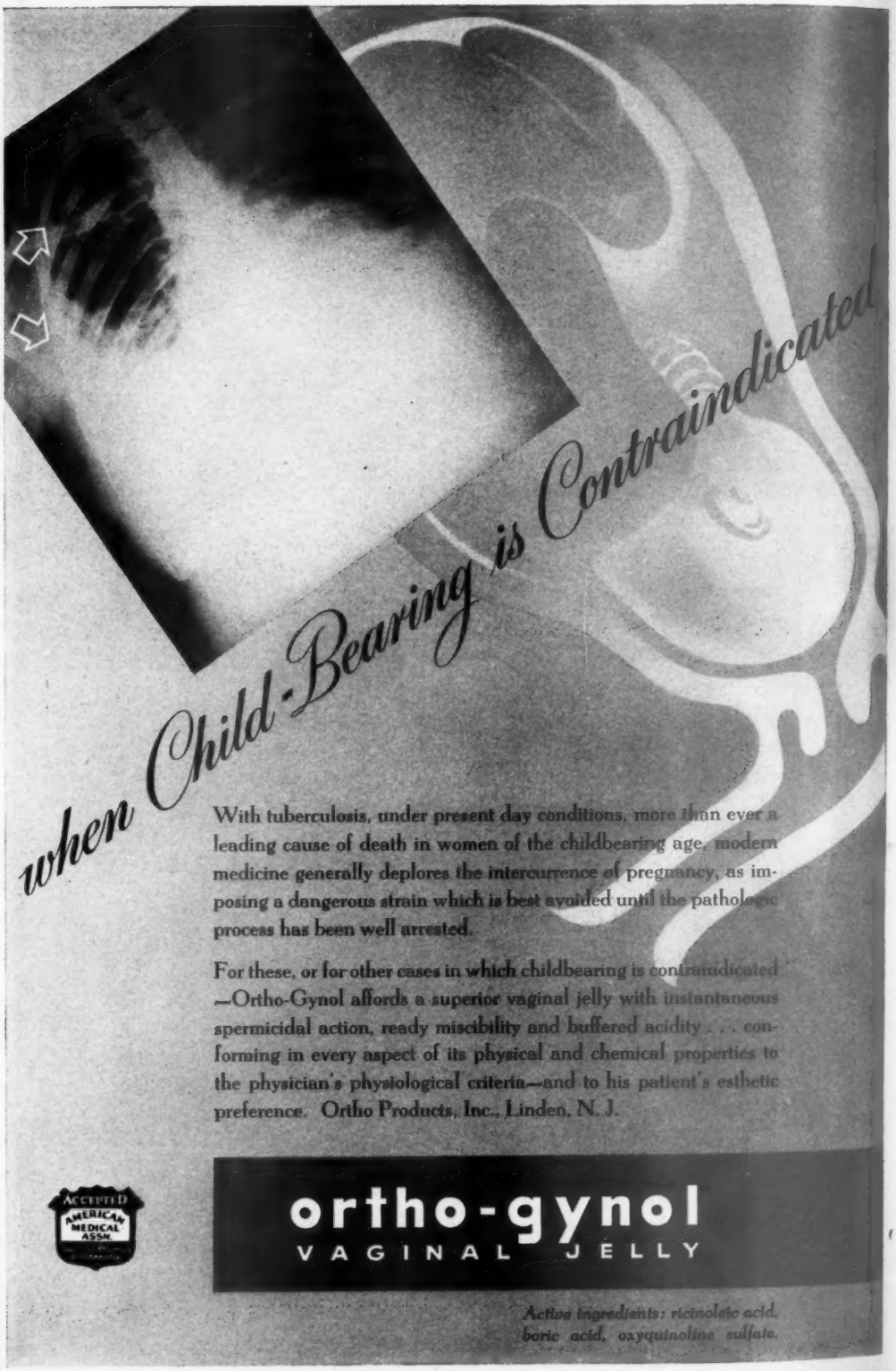

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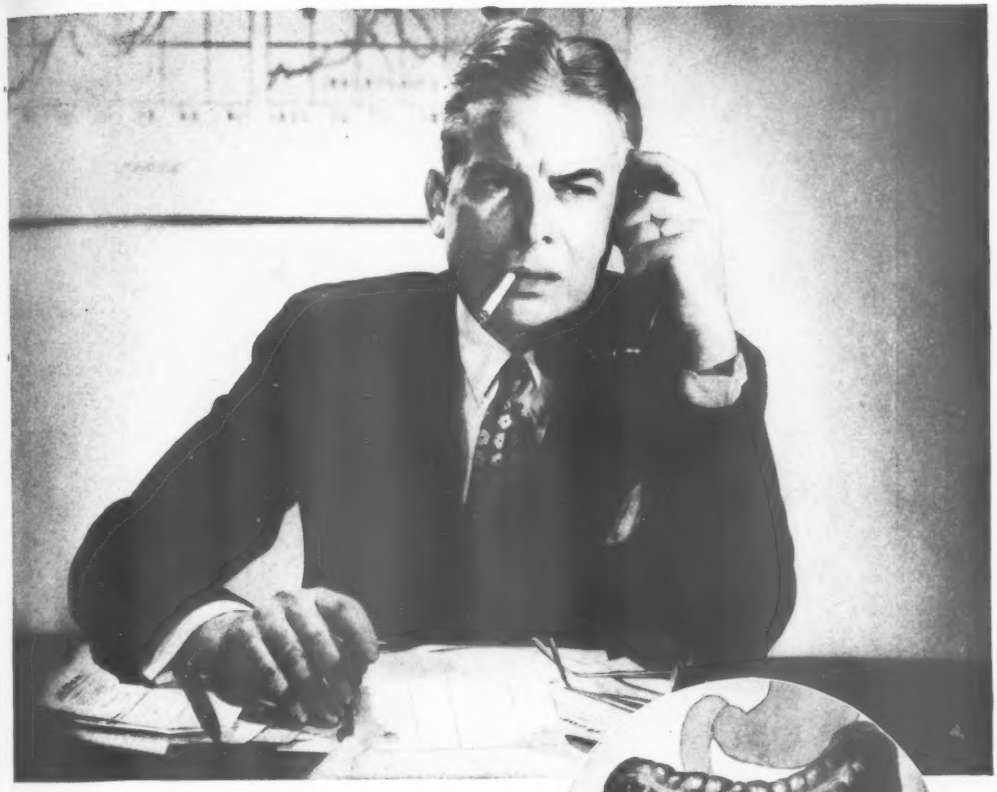

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